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Editorial

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TO ERR IS human. This commonplace saying is so well established in our everyday thinking and speech that we ordinarily take it for granted and behave accordingly. It is difficult for most of us to become indignant about the death toll on our highways even though it is a constant grisly reminder of human imperfection. Insurance companies tabulate, predict, and capitalize on our blunders. Teachers make their living because humans err. We even use errors in evaluating the performance of our favorite baseball team.

Acknowledging this human weakness does not mean that we must adopt an attitude of fatal resignation toward it. Banks rarely make mistakes, not because they are staffed by superhuman individuals, but because they have set up such an intricate series of balances and checks that errors seldom go undetected. It is an extremely rare thing to find drugs improperly labeled, not because pharmaceutical houses have dispensed with human operators, but because of the elaborate checks and counterchecks which control their operations. Safety engineers in industry sometimes achieve superlative safety records, not by placidly accepting human failures, but rather by designing operations and the work environment to eliminate error-provocative situations.

No one likes to admit that he has made an error. When errors involve the highly ethical and emotionally charged problems of the hospitalized patient, they become doubly difficult to study. Perhaps for these reasons errors in medical practice have rarely been subjected to scientific inquiry. But doctors and nurses

are people, too, and it would be unreasonable to expect that the everyday business of hospital routine is carried out infallibly. If errors do occur, and if we want to do something about improving the situation, we must allow it to become the subject of legitimate scientific study.

A few years ago The Johns Hopkins Hospital set up an Operations Research Program to study administrative practices and operations in the hospital using some of the newer methods of industrial engineering and operations research. As part of this program, and with the splendid cooperation of the supervisory and nursing staff, we were fortunate in being able to set up and conduct a study of medication errors in the hospital. The method we used was one which has been successfully applied to the investigation of accidents and near accidents in aviation—the critical incident technique. This is a procedure for collecting direct observations of complex human behavior. It is specifically aimed at the solution of practical problems because it attempts to provide insight into methods of correcting potentially dangerous situations.

To begin our research the experimenter spent several weeks becoming completely familiar with the medication system by observing it carefully in the company of various nursing personnel. Next the experimenter and the director of nursing service studied 250 medication incidents to find out some of the things that had occurred in the past and to get some insight into what might be expected in this investigation. Medication incidents are written up and filed with accounts of all unusual occurrences in the treatment and care of patients and are kept primarily for administrative and legal reasons. The practice of writing up such incidents at the time they occur is part of a long-established routine at the Hopkins Hospital and is taught as a required procedure to all nursing personnel. Careful study of these incidents revealed a number of common features and suggested several common causes underlying their occurrence.

The next step involved the design of a questionnaire for collecting medication incidents as they occurred. The requirements we kept in mind in designing this questionnaire were: (a) Nursing personnel were to fill out a questionnaire whenever they made a medication error or near error or saw someone elso do so; (b) respondents were to remain anonymous to encourage participation; (c) the questionnaire had to be brief yet should contain all items relevant to the study; and (d) it should serve the dual purpose of collecting and categorizing incidents.

Getting the study under way required a special orientation program. The experimenter first met with the supervisory personnel of all the clinics of the hospital. This was followed by a series of interviews with the nurses of each division. In these orientation talks the experimenter described the purpose of the study, the questionnaire, and the procedure. The nurses were told that they were to fill out a questionnaire as soon as possible after a medication error or near error occurred, place it in an envelope, seal it, and leave it in one of the boxes which would be located on every floor where medications were administered. Instructions especially emphasized that all reports would be anonymous. Because the data collection extended over a period of seven months it was necessary to conduct additional orientation sessions from time to time to acquaint new nurses and students with the program.

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Over a period of seven months we collected 178 medication incidents. Of the total number, 80 per cent (143) were medication errors and 20 per cent (35) were near errors. Although the sample of near errors was small, there appeared to be no outstanding differences between them and the actual errors. (This is in accord with the general findings of industrial accident studies which show that it is largely a matter of chance whether a particular incident will result in an accident or near accident.) It was gratifying to note that, despite their inconvenience, the questionnaires were apparently not treated in a casual or perfunctory manner. Perhaps the best evidence for this is the fact that, with few exceptions, the respondents took the time and trouble to add amplifying statements and remarks. Further evidence of the interest generated in the study is that some nurses, on their own initiative, compiled lists of frequently confused drugs, saved samples of poorly labeled medicine containers, and so on.

The analysis of the data was aimed not only at focusing attention on the problems of the medication system but also at suggestions for instituting corrective action. The incidents were classified in six different ways: (1) by type of error and, within each type, by cause; (2) by cause without reference to type of error; (3) by job classification of the person who made the error; (4) by clinic of the hospital where the incident occurred; (5) by time of day; and (6) by patient load.

Below in highly abbreviated form are shown the major headings into which the 178 incidents could all be grouped.

TYPE OF INCIDENT	NO. REPORTED
1. The *wrong patient* received or almost received a medication	36
2. A patient received or almost received a *wrong dose* of medication	36
3. A patient received or almost received an *extra (unordered) dose* of medication	36
4. A patient's medicine was *omitted* or almost omitted	31
5. A patient received or almost received the *wrong drug*	23
6. A patient received or almost received medication at the *wrong time*	14
7. A patient received or almost received the medicine through an *improper route*	2

An analysis of the reasons given for the incidents shows that 90 per cent (169 of 187) of them are contained within five categories: (1) Failure to follow required checking procedures; (2) misreading or misunderstanding written communications; (3) transcription errors; (4) medicine tickets misfiled in ticket box; and (5) calculational errors.

Observations and interviews at the start of the study showed that certain clinics, generally the ones with large concentrations of student nurses, were under greater pressure and stress than others. Student nurses, of course, acquire some of their training on the wards and this of necessity means that they are not as thoroughly practiced in medication procedures as are regular nurses. For these reasons, it seemed worth while to study the incidents according to the clinic where they occurred and the job classification of the respondent. The data

confirmed that most of the incidents were reported by student nurses and that all but 16 of the incidents occurred in 4 of the 8 clinics. These clinics are the ones in which the heaviest loads of medicines are administered or in which the nursing personnel are under stress because the clinic contains psychiatric patients who are extremely difficult to handle. Although most of the incidents were reported by student nurses, the proportion of incidents reported by the student nurses in each of the 4 clinics agrees generally with the proportion of the medications which they administered in each clinic. This close correspondence between the proportion of medicines given by student nurses and the proportion of incidents they contributed suggests that the job classification is not as important a variable as the medication load or other factors associated with a particular clinic.

An analysis of the incidents according to the time of day in which they occurred showed several peaks. The most pronounced one occurred at 10:00 A.M. with smaller distinct ones at 8:00 A.M., 12:00 noon, 2:00 P.M., 4:00 P.M., 5:00 P.M., and at 10:00 P.M. These findings are explained by one or more of the following factors:

Preferred Times for Scheduling Medications.—Medicines to be given once a day are often scheduled for 10:00 A.M. Medications scheduled for three times a day are often administered at 10:00 A.M., 4:00 P.M., and 10:00 P.M.; or 8:00 A.M., 2:00 P.M., and 8:00 P.M.

Stress Periods.—In some wards 10:00 A.M. is a period of high stress. A large number of medications must be administered while nurses are still completing their morning chores. This is also the time at which doctors customarily make their ward rounds so that there is a heavy concentration of staff on the floor.

Meal Times.—During meal hours there is often only one nurse on the floor to administer all the medications. Furthermore, student nurses are sometimes assigned to unfamiliar wards to relieve the regular staff during meal hours.

Change of Shift.—The change of shifts at 8:00 A.M. and 4:00 P.M. creates a definite break in continuity with the result that oncoming nurses are often times incompletely briefed.

An unexpected finding was that the greatest number of incidents was reported when a nurse administered medication to only one patient. This finding was on the face of it so unreasonable that it received careful investigation. The explanation appears to be as follows: Nurses are rarely scheduled to administer medication to only a single patient at a time. Single patient assignments almost invariably represent unscheduled orders which are to be carried out immediately. One of the two major kinds of unscheduled orders is a *stat.* medication. When the nurse is given such an order she must stop what she is doing, copy the order onto a medicine ticket, prepare the drug, administer it, and sign the order off on the appropriate forms. The other major type of unscheduled order is a *preoperative* order. Even though a patient may be scheduled for an operation at a certain hour and the medication order written for that time, a nurse is not assigned to execute the order until the call comes from the operating room that the doctors are ready to receive the patient. The call may come earlier or later than the scheduled time. When it comes the nurse must stop what she is doing, carry out

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the order, and take the patient to the operating room. Since either type of order interrupts her normal routine the nurse is more likely to forget to follow procedures and, in so doing, commit an error.

Perhaps the most important part of our findings concerns recommendations for the design of a safer and more efficient medication system. These are grouped under four major headings: (1) recommendations dealing with written communication; (2) recommendations dealing with medication procedures; (3) recommendations dealing with the working environment; and (4) recommendations for training and education.

Our analysis of the incidents collected in this study and of the causes contributing to these incidents shows that one of the major difficulties with modern medication procedures revolves around the general problem of written communication. This is a problem which plagues the business man, the executive, the labor leader, and is involved in almost every aspect of our modern civilization. In hospital work drugs with similar names but different usages, confusing nomenclatures, illegible handwriting, drug labels which are so small as to be scarcely visible, and abbreviations which resemble each other are all examples of poor written communication which almost literally invite human error. For this reason, the first and most important series of recommendations to originate from our study has to do with this extremely serious problem in hospital practice. We feel, in particular, that much could be done to improve the system of drug nomenclature. In many cases in which a nurse misread a label, she selected a drug with a name similar to the one ordered. When she made an error in copying the name of the drug ordered by the doctor onto a nursing care card and medicine ticket she often wrote the name of a drug similar to the one ordered. In addition, nurses reported that they were sometimes confused by the fact that there are many drugs of the same chemical composition but with different trade names. Instances such as these point to the fact that the present system of drug nomenclature is confusing and can stand much improvement.*

Other deficiencies dealing generally with the problem of communication are: (1) the method of writing drug dosages (Nurses oftentimes failed to note decimal points so that 2.5 mg. was read as 25 mg. or 0.1 c.c. read as 1 c.c.); (2) confusions arising from standard medical terminology and abbreviations (milligrams [mg.] was confused with micrograms [mcg.]; "every night" [q.n.] with "every hour" [q.h.]; and so on); (3) the visibility of medication orders and handwriting in general; (4) the legibility of drug labels; and (5) the arrangement of drugs in the ward pharmacy.

To attack errors which occurred because nurses did not follow correct medication procedures we suggest that difficulties associated with particular procedures be singled out and that changes be made so that the procedures are easier to follow. In addition, we suggest that more safety checks and redundancy be built into medication procedures so that if a nurse should forget to follow one step or make an error it would be caught by some other step. One way of building

*For example here are some drug names which were confused with each other: digitoxin and digoxin, phenobarbital and pentobarbital.

such additional redundancy into medication procedures is to provide the patient himself with a little more information about the medication he is scheduled to receive. Being vitally concerned with his own health and safety, he would thus become an active and interested partner in eliminating certain kinds of common errors. The patient who knows, for example, that he is supposed to receive two blue pills every four hours (even though he may not know what they are supposed to do) will almost certainly object if a nurse attempts to give him an intramuscular injection, if he is offered two yellow pills, or if he is given two blue pills only fifteen minutes after having already swallowed two.

As regards the general working environment, we find that a substantial number of nurses committed errors because they were distracted or interrupted in their work. This suggests that the environment in which medication orders are processed and carried out should be carefully studied and modified so that the nurse can work in a more nearly optimal environment.

Our final recommendation concerns the improvement of the system by seeing to it that nursing personnel are well trained in medication procedures, alerted to the hazards of their work, and highly motivated to avoid error. We think, in particular, that the instructional staff of a hospital, instructors in the classroom, and head nurses on the wards should make use of incidents such as we have collected to dramatize some of the kinds of errors which can and do occur and some of the places in which extra care must be taken to avoid their occurrence.

Our general conclusion is that nurses are indeed people, and being people, they make mistakes. Fortunately, however, people also have some virtues. They are self-evaluative, critical, and inventive. Given sufficient time and effort we believe that these constructive abilities can be used to devise more nearly foolproof solutions to the problem of medication errors.

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STUDIES OF ALCAPTONURIA: CONTENT AND DENSITY OF THE WATER AND SOLID PHASES OF OCHRONOTIC CARTILAGE

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INTRODUCTION

IT has long been observed that alcaptonuric individuals characteristically and in temporal sequence develop homogentisic aciduria (alcaptonuria)[1], then ochronosis,[2,3] and finally degenerative joint disease.[4,5]

Early in the course of the disease, individuals homozygous for the rare autosomal recessive allele[6,7] present only homogentisic aciduria, the first of the three distinct components of the clinical syndrome. Subsequently, connective tissue of the skeletal, integumentary, ocular, and cardiovascular systems tends to become pathologically pigmented, and thus to present the characteristic features of the second component of the complete syndrome, ochronosis. Finally, the third and clinically most distressing component of the triad, alcaptonuric (ochronotic) arthritis supervenes. Usually this occurs at an age significantly earlier than arthritis in the general population. Arthritic manifestations appear initially in the axial skeleton, especially in the lower lumbar region, but later appear in the joints of both the axial and appendicular skeleton. The tissue process, other than the pigmentation, appears similar to that of "senile" osteoarthritis.

Curiously, despite the obvious implications of the clinical observations of the syndrome in which many biochemical facets have been well elucidated, only passing attention appears to have been directed to the chemical anatomy of affected tissues. Accordingly, during the course of experimental studies on the pathogenesis of alcaptonuric arthritis in this laboratory, costal cartilage was obtained from a patient with ochronosis and an attempt was made to characterize

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some of its properties. The present report is concerned with but one of these studies, specifically the content and density of the water and solid phases of a segment of ochronotic cartilage.

MATERIAL AND METHODS

One alcaptonuric subject with both ochronosis and arthritis (Figs. 1 and 2) and 6 nonalcaptonuric control patients served as the source of the cartilage specimens. Clinical and laboratory examinations of the alcaptonuric subject are presented in detail elsewhere.[8]

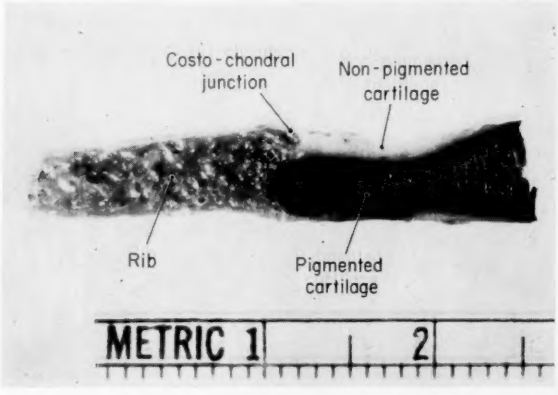

Fig. 1.—Costochondral junction of the alcaptonuric subject (R. S.). The central portions of the costal cartilage are densely pigmented, whereas the peripheral portions are relatively nonpigmented. Bone is entirely devoid of pigmentation.

A portion of costal cartilage of full thickness just distal to the costochondral junction was obtained from each of these subjects either at operation or at necropsy. The specimens were freed from perichondrium, bone and calcified cartilage and aliquots were placed in prechilled flasks of water and of physiologic saline. (One portion of a cartilage specimen was placed in water and another aliquot in saline.) Temperature was maintained at 4° C. Tissue obtained at operation was prepared within 30 minutes of surgical exposure of the region; material from autopsies was obtained within 5 hours of the patient's demise.

Representative aliquots of each cartilage specimen were weighed in water and saline after a 2 to 4 hour period of equilibration at 4° C. Then, still fully hydrated, they were weighed in air and then weighed again submerged either in water or saline. The weight in air represented the mass of the hydrated specimen (hydrated mass whole tissue) and the weight in either water or saline represented the volume of the hydrated specimen (hydrated volume whole tissue).

All specimens were then dried in air at 50° C. for 96 hours or longer, if constant weight had not been obtained at that time. (In general, when a specimen failed to show a percent weight change of ± 1.0 to 1.5 per cent over its previous weight determination, constant weight appeared to have been achieved.) Having been dried in air to constant weight at 50° C., the specimens were then dried in

air at 100 to 105° C. to constant weight. Finally the specimens were burned to a white ash at 600° C. Data for the mass, volume, and density of the water and solid phases were calculated as described previously.[9]

RESULTS

Table I summarizes clinical data concerning the 7 study patients. Representative data from 17 different specimens of cartilage obtained from one patient (F. D.) are presented in Table II to indicate the reproducibility of the analytical technique. In Tables III and IV pertinent data are presented on the content and density of the water and solid phases of cartilages obtained from 5 of the remaining 6 patients. All of the data on dry material has been calculated using the constant weight values obtained on drying the specimens at 105° C. Data on the cartilage specimen of the seventh patient are included in Fig. 3 which is a semilogarithmic plot of the percentage of the specimen's fully hydrated weight remaining (i.e., rate of loss of water content) after drying for various times at 100 to 105° C.

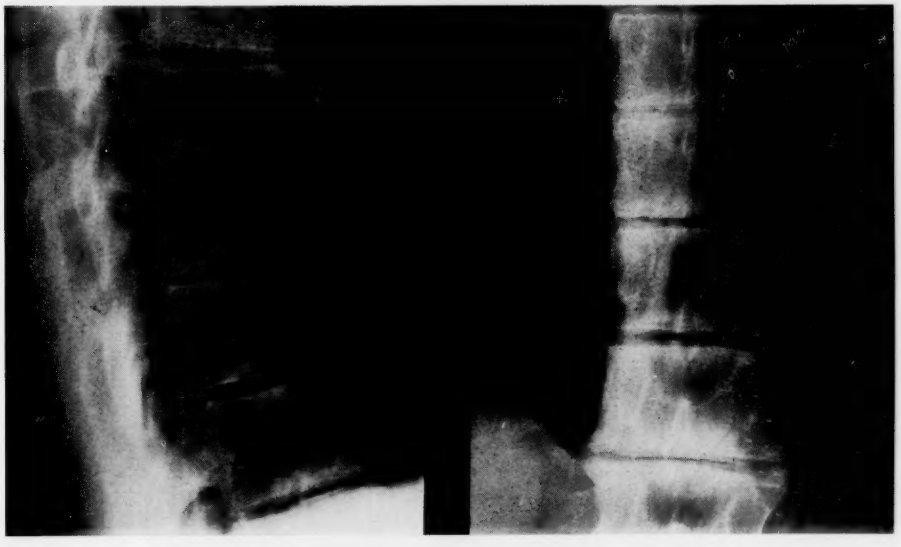

Fig. 2.—Antero-posterior and lateral roentgenograms of the spine of the study patient (R. S.) at age 34. Pronounced degenerative changes as well as intervertebral disc calcification, frequently considered a pathognomonic roentgen sign of alcaptonuria, are readily apparent in both views. "Vacuum signs" are prominent in the upper two intervertebral disc spaces, especially on the lateral view.

DISCUSSION

When the weight loss on drying at 50° C. is plotted against time on semilogarithmic coordinates (Fig. 3), it is apparent that the dehydration process involves at least two distinct rate processes; a rapid and a slow component. In all of the present study specimens, once a zero rate of change on the slow com-

ponent was obtained on drying at 50° C., no further significant change was observed on additional heating at 105° C. This would suggest that at least two, more or less, distinct water compartments are being measured; one, represented by values obtained on drying at 50° C. over a period of about 12 hours, and a second, represented by data obtained thereafter by heating both at 50 and 105° C. Since equilibrium weight values after drying at 100° C. were consistently lower than those obtained on drying at 50° C. by a factor of 1.5 per cent or less in almost all instances in both the normal and the ochronotic cartilage specimens, the data would suggest that the differences are probably not significant and that the shrinkage temperature of collagen may not be either limiting or critical in this determination.

Of particular interest, moreover, was the observation that the specimens fell into two more or less distinct groups dependent apparently solely on age (Table IV). This is in accord with previous animal data in the literature.[10]

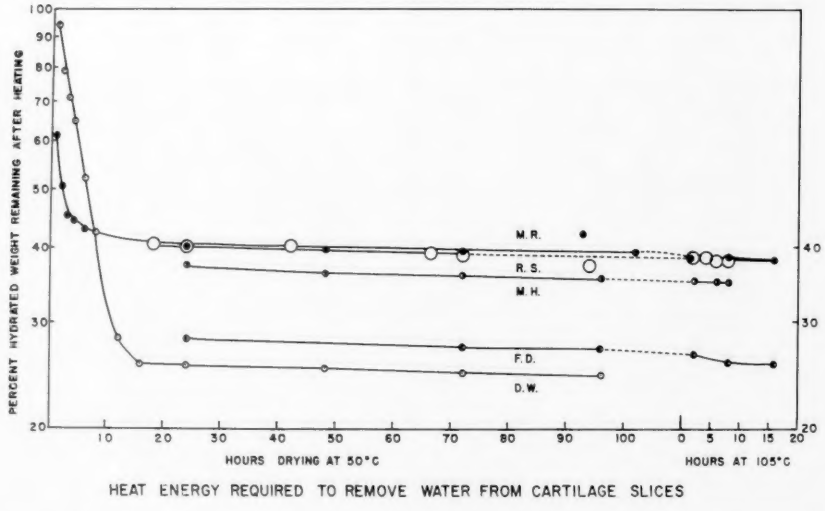

Fig. 3.—Semilogarithmic plot of the rate of water loss of cartilage specimens on drying to indicate the heat energy required to remove water. The data are presented in terms of the percentage of the initial hydrated weight remaining after drying at 50° and 105° C. for the indicated time intervals expressed in hours.

Unfortunately, owing to the necessity of obtaining only a small amount of biopsy material, no separate analysis could be made between the obviously pigmented and the apparently nonpigmented regions of the ochronotic cartilage specimen (Fig. 1). The fact, however, that the organic, inorganic, and water phases of the whole ochronotic specimen were practically identical to that of the control specimen from a patient of similar age suggests the obvious likelihood that relatively small chemical changes were, in fact, responsible for the very striking gross appearance of the ochronotic specimen. Indeed, had quite a large

mass of homogentisic acid, or any of its polymers, been deposited in the ochronotic cartilage it might have been expected that correspondingly large changes would have been observed in the magnitude of the solid phase of the ochronotic specimen and that these might have been distinctly different from the control specimens. This, however, was not observed, nor were there any significant alterations in the magnitude of the water phase of the specimen.

Accordingly, if the present data are interpreted as being representative of hyaline cartilage elsewhere in the body of the alcaptonuric patient, it would appear that the gross chemical anatomy of hyaline cartilage in alcaptonuric subjects does not significantly differ from that of the normal. Presumably, therefore, the observed pathologic changes in the intervertebral discs of this patient, as in other patients with the alcaptonuria syndrome, must arise in consequence of some more subtle chemical alterations.

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TABLE I. CLINICAL DATA ON SEVEN PATIENTS FROM WHOM CARTILAGE SPECIMENS WERE OBTAINED

NAME	HISTORY NO.	AGE (YRS.)	SEX	DIAGNOSIS	SOURCE OF SPECIMEN
M.H.	24–58–32 (A–28736)	75	F	Pulmonary Embolus	Autopsy
R.S.	76–12–70	34	M	Alcaptonuria	Operation
M.R.	65–43–03	30	F	Atrial Septal Defect Pulmonary Stenosis	Operation
A.T.	46–08–64	15	F	Atrial Septal Defect Pulmonary Stenosis	Operation
F.D.	83–79–64	10	M	Pectus Excavatum	Operation
D.W.	85–20–04	6	M	Pectus Excavatum	Operation
P.P.	B–57131 (A–28737)	½	M	Atrial Septal Defect Pulmonary Stenosis	Autopsy

The very striking relative paucity of cells in ochronotic cartilage as compared with the normal[11] may yield some clue in this regard and is currently under active investigation. It is possible, since homogentisic acid polymers have been found to tan collagen in vitro,[12] that a tanning phenomenon may occur in vivo. So far as the present data are concerned, however, it would seem that neither displacement of chondroitin sulfate in association with water, as observed in other conditions,[13] nor the addition of water to collagen are likely to be involved, since there were no significant alterations in the content and density of the water and solid phases of the ochronotic cartilage specimen.

SUMMARY AND CONCLUSIONS

The content and density of the solid and water phases of a biopsy specimen of ochronotic cartilage have been studied in an attempt to account for some of the pathologic findings in ochronosis and alcaptonuric arthritis. It was found that the gross chemical anatomy of the whole ochronotic cartilage biopsy speci-

TABLE II. REPRESENTATIVE DATA ON 17 CARTILAGE SPECIMENS OBTAINED FROM CONTROL SUBJECT F. D.

SPEC. NO.	HYD. MASS (MGS.)	HYD. VOL. (MLS.)	HYD. DENSITY	PERCENT HYDRATED MASS			PERCENT HYDRATED VOLUME			DENSITY	
				WATER	ORGANIC	INORGANIC	WATER	ORGANIC	INORGANIC	ORGANIC	SOLID
W* 1	654	599	1.09	74.3	24.3	1.4	81.5	18.0	0.5	1.47	1.51
W 2	828	756	1.10	71.6	27.1	1.3	78.7	20.8	0.5	1.43	1.46
W 3	342	316	1.08	76.0	22.5	1.5	82.6	16.8	0.6	1.45	1.49
W 4	320	299	1.07	75.6	23.4	1.0	81.3	18.4	0.3	1.36	1.39
W 5	332	304	1.09	73.8	25.0	1.2	80.9	18.8	0.3	1.46	1.50
W 6	219	199	1.10	73.1	25.6	1.3	80.4	19.1	0.5	1.47	1.51
W 7	231	212	1.09	71.9	26.8	1.3	78.3	21.2	0.5	1.38	1.41
W 8	235	214	1.10	73.6	25.1	1.3	80.8	18.7	0.5	1.47	1.51
W 9	226	207	1.09	76.6	22.1	1.3	83.6	15.9	0.5	1.51	1.56
S† 1	843	771	1.09	73.7	24.8	1.5	80.8	18.7	0.5	1.45	1.50
S 2	912	836	1.09	73.2	25.0	1.8	80.1	19.3	0.6	1.42	1.47
S 3	356	321	1.11	70.8	28.1	1.1	78.8	20.9	0.3	1.49	1.53
S 4	271	245	1.11	73.8	24.7	1.5	81.6	18.0	0.4	1.52	1.58
S 5	291	266	1.09	73.9	25.4	0.7	81.2	18.4	0.4	1.51	1.52
S 6	211	190	1.11	71.6	27.0	1.4	79.5	20.0	0.5	1.50	1.54
S 7	146	132	1.11	74.7	23.3	2.0	82.6	16.7	0.7	1.54	1.61
S 8	163	147	1.11	73.0	25.2	1.8	80.9	18.4	0.7	1.52	1.57

*Weighed in saline.
†Weighed in water.

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TABLE III. DATA ON THE CONTENT AND DENSITY OF THE WATER AND SOLID PHASE OF THE CARTILAGE SPECIMENS IN SIX PATIENTS

NAME	AGE (YRS.)	SPEC. NO.	WHOLE TISSUE			WATER PHASE				SOLID PHASE					ORGANIC PHASE					INORGANIC PHASE			
			HYD. MASS (MGS.)	HYD. VOL. (MLS.)	HYD. DENSITY	MASS (MGS.)	MASS (%)	VOL. (MLS.)	VOL. (%)	MASS (MGS.)	MASS (%)	VOL. (MLS.)	VOL. (%)	DENSITY	MASS (MGS.)	MASS (%)	VOL. (MLS.)	VOL. (%)	DENSITY	MASS (MGS.)	MASS (%)	VOL. (MLS.)	VOL. (%)
M.H. (24-58-32)	75	W 1*	384	343	1.12	271	70.6	271	79.0	113	29.4	72	21.0	1.57	94	24.5	66	19.2	1.42	19	4.9	6	1.8
		W 2	245	219	1.12	159	64.9	159	72.6	86	35.1	60	27.4	1.43	85	34.7	59.7	27.3	1.42	1	0.4	0.3	0.1
		S† 1	211	178	1.19	128	60.7	128	71.9	83	39.3	50	28.1	1.66	68	32.2	45	25.3	1.51	15	7.1	5	2.8
		S 2	96	84	1.14	62	64.6	62	73.8	34	35.4	22	26.2	1.55	33	34.4	21.7	25.8	1.52	1	1.0	0.3	0.4
R.S. (76-12-70)	35	W 1	538	474	1.14	334	62.1	335	70.7	204	37.9	139	29.3	1.47	195	36.2	136	28.7	1.43	9	1.7	3	0.6
		S 1	558	490	1.14	346	62.1	347	70.8	211	37.8	143	29.2	1.48	200	35.9	139	28.4	1.44	11	2.0	4	0.8
M.R. (65-43-03)	30	W 1	450	395	1.14	280	62.2	280	70.9	170	37.8	115	29.1	1.48	161	35.8	112	28.3	1.44	9	2.0	3	0.8
		S 1	416	364	1.14	257	61.8	257	70.6	159	38.2	107	29.4	1.49	151	36.3	104	28.6	1.45	8	1.9	3	0.8
A.T. (46-08-64)	15	W 1	166	151	1.10	123	74.1	123	81.5	43	25.9	28	18.5	1.54	40.7	24.5	27.2	18.0	1.50	2.3	1.4	0.8	0.5
		W 2	100	92	1.09	74	74.0	74	80.4	26	26.0	18	19.6	1.44	24.9	24.9	17.6	19.1	1.41	1.1	1.1	0.4	0.5
		W 3	83	76	1.09	61	73.5	61	80.3	22	26.5	15	19.7	1.47	20.3	24.5	14.4	18.9	1.41	1.7	2.0	0.6	0.8
		S 1	71	65	1.09	53	74.7	53	81.5	18	25.3	12	18.5	1.50	16.7	23.5	11.6	17.9	1.40	1.3	1.8	0.4	0.6
		S 2	74	68	1.09	56	75.7	56	82.4	18	24.3	12	17.6	1.50	17.1	23.1	11.7	17.2	1.46	0.9	1.2	0.3	0.4
		S 3	34	31	1.10	25	73.5	25	80.6	9	26.5	6	19.4	1.50	8.3	24.4	5.8	18.7	1.43	0.7	2.1	0.2	0.7
P.P. (B-57131)	½	W 1	105	97	1.08	84	80.0	84	86.6	21	20.0	13	13.4	1.62	20.2	19.2	12.7	13.1	1.59	0.8	0.8	0.3	0.3
		W 2	91	85	1.07	73	80.2	73	85.9	18	19.8	12	14.1	1.50	17.4	19.1	11.8	13.9	1.47	0.6	0.7	0.2	0.2
		S 1	66	61	1.08	53	80.3	53	86.9	13	19.7	8	13.1	1.63	12.3	18.6	7.8	12.8	1.58	0.7	1.1	0.2	0.3
		S 2	60	56	1.07	48	80.0	48	85.7	12	20.0	8	14.3	1.50	11.8	19.7	7.9	14.1	1.49	0.2	0.3	0.1	0.2

*Weighed in saline.
†Weighed in water.

TABLE IV. SUMMARY OF THE AGE EFFECT ON THE MAGNITUDE OF THE WATER AND SOLID PHASES OF CARTILAGE SPECIMENS IN COMPARISON WITH DATA FROM ANIMALS

	HUMAN COSTAL		PUPPY COSTAL	PUPPY ARTICULAR
Total water (Gms./kgm.)	adult children	636 766	745	764
Total solids (Gms./kgm.)	adult children	363 234	254	236

men was entirely normal, though histologic examination revealed a significant paucity of cartilage cells per unit area. It is concluded that the striking gross changes seen in ochronotic cartilage probably arise as a consequence of quite subtle chemical alterations, which were entirely undetected by the methods employed here, and that chondroitin sulfate and water displacement, as observed in other affections of the intervertebral discs, are in all probability not involved.

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Therapeutics

RADIOTHERAPY OF LETHAL MID-LINE GRANULOMA

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IN December, 1896, Dr. Peter McBride[23] presented to the Laryngological Society of London, clinical photographs of "a case of rapid destruction of the nose and face." In 1933, Stewart[30] reported on 10 cases, personally observed or culled from the literature, of what he termed "Progressive Lethal Granulomatous Ulceration of the Nose." Since that time many other cases have been described under different names, and in the more recent literature the syndrome has been described variously as lethal mid-line granuloma, malignant granuloma, or as granuloma gangraenescens. There seems little doubt that the entities described under these varying names all refer to an identical condition. Some one hundred case reports have appeared in the literature but unfortunately full clinical and pathologic descriptions are not always given and the diagnosis must remain in some doubt in several of the cases.

ETIOLOGY

The condition is much more common in men than in women, probably in the ratio of eight or nine to one. Although cases in individuals over 70 and below 20 have been reported, it occurs most frequently in the fourth decade. In the present series the average age was somewhat older (Table I). Nothing is known of the causation of the disease. No bacterial agent has been consistently identified, no virus or fungus isolated, and careful study of one of our cases by electron microscopy and virus cultures failed to reveal any evidence of an infective agent. Apart from the fatal outcome, the clinical and pathologic findings are not those of a malignant disease. A resemblance to Wegener's granulomatosis has been noted by several authors and some even believe that the two conditions are identical. There seems general agreement that Wegener's granulomatosis is a hypersensitive response to an unknown allergen and on the basis of the sug-

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gested kinship of the two diseases, steroids have been used with temporary benefit in some cases of lethal mid-line granuloma.[17,32,33] In the absence of histologic identity, the evidence is in our view insufficient to postulate identity on the basis of similar clinical appearance and response to therapy. It is our feeling that the two conditions should be separated.

TABLE I. CHARACTERISTICS OF PATIENTS WITH MID-LINE GRANULOMA AT JOHNS HOPKINS HOSPITAL

CASE	SEX	AGE	DURATION	SITE
1	M	60	15 years	Septum
2	M	45	5 years	Right side of nose
3	M	33	1 year	Left side of nose
4	M	33	7 years	Right side of nose
5	F	73	18 years	Antrum
6	M	74	6 months	Palate
7	F	21	6 months	Septum

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CLINICAL COURSE

Little can be added to the masterly description of the clinical picture given by Stewart[30] in his paper in 1933. Essentially, he noted three stages in the disease: (1) a prodromal phase of nasal stuffiness unrelieved by the usual measures, this lasting sometimes for several years; (2) the phase of obstruction, foul-smelling discharge with spread to skin and neighboring structures, crusting and sequestration of cartilage, fever, and hemorrhage (notably absent in this phase was the normal physiologic response of leukocytosis and there was a remarkable absence of pain); and (3) death from exhaustion and terminal hemorrhage about one year from the onset of the active phase.

Stewart did not observe any evidence of systemic involvement in his cases but such manifestations may occur. With more modern methods of radiologic examination, intrapulmonary disease has been noted in several cases and, at autopsy, involvement of many other vital organs has been demonstrated.[29] The skin is not infrequently involved in areas distant to the original site of disease (see Cases 4 and 5), but the relentless massive destruction of the facial tissues remains the most prominent feature of the condition.

PATHOLOGY

A vivid description of the destructive process was given by O'Sullivan in a case reported by Sir Robert Woods[35] in 1921, "It seemed as if a wave of granulation tissue advanced irregularly into the healthy parts, breaking down behind as it advanced in front." Detailed pathologic observations are recorded by Friedmann[9] and by Hultberg and co-workers.[17] In 1956, Spear and Walker[29] reported the findings in Case 4 of the present series. In this latter paper, the authors differentiate Wegener's granulomatosis from lethal mid-line granuloma. They contrast the intense arteritis with occasional polymorphonuclear reaction observed by Wegener with the bland vascular necrosis unaccompanied by inflammatory

reaction found in most of the lesions in their study. Furthermore, a full autopsy failed to reveal glomerulonephritis, generalized periarteritis, or large numbers of foreign body and Langhans-type giant cells which Wegener characterized as being present in the condition associated with his name. It is interesting to note that Wegener himself[31] differentiated between the two conditions.

TREATMENT

The number of treatments advocated corresponds with the number of postulated causative factors. Thus, antisyphilitic treatment was used in several cases in the earlier literature.[23,30,35] With the advent of sulfonamides and occasionally antibiotics, intensive therapy directed at the supposed infective agent led to some clearing of the process, and Sneddon and Colquhoun[27] reported a case with survival for one year after the administration of chloramphenicol. The response of Wegener's granuloma to ACTH and cortisone, and a thoughtful and well-argued paper by Williams,[32] led to the exhibition of these steroid drugs in many cases, often with temporary control of the disease.[17,32,34] However, sometimes these drugs were without effect.[1,26] Surgical removal was tried by Stewart[30] (who found diathermy useless), by Bayer,[2] and by Rasmussen,[25] who recommended radical removal with postoperative radiation. Edgerton and DesPrez[5] believe that radical surgery is the treatment of choice and state that radiation therapy has little or nothing to offer.

Radiation therapy has been employed in many cases in the past, the earliest recorded being that of M'Arthur and Dew.[22] Improvement was noted in this case, in 3 of those reported by Stewart,[30] and in cases reported by Hoover,[13] Isbell,[18] and Williams.[32] No change in the course of the disease was noted after the administration of radiation therapy to those patients reported by Simson Hall,[11] Lewy,[20] Woodburn and Harris,[34] Lierle,[21] Rasmussen,[25] and Levan.[19] Many of these cases were treated prior to modern dosimetry, in others no dosage measurements are given, and from such expressions as "radium emanations were used," "single full doses of high frequency x-ray," "one-third pastille dose," little idea of the dosage delivered to the lesion can be gained. Not overmuch credence should, therefore, be given to the results obtained.

In 1938, Heinermann,[12] reported healing of an extensive ulceration of the face for one year by delivering 150 r daily to each of three fields of 10 by 15 cm. at 180 KV to a total of 2,500 r. Sneddon and Colquhoun[27] gave 2 doses of superficial x-ray therapy, the first 200 r producing some improvement and the second dose, of 100 r, healing the lesion, but antibiotics were given concurrently and chloramphenicol was credited with the temporary success. In the case reported by Pardo-Castello and associates,[24] ulceration which had been relentlessly progressive for 9 months improved for 8 months after 600 r was given on alternate days to a total of 3,000 r, but where this dosage was measured and through what fields it was given are not stated. Bergqvist and Koch[4] reviewed the literature relating to radiation therapy and reported a case treated by small doses of radiation with no effect, but which had a rapid response in the areas of the local lesion to doses of 500 r on 8 successive days. They recommended radiotherapy in "tumor doses." A further review by Friedmann[9] of 15 cases personally studied

showed that 7 patients were dead, one of whom had received x-ray therapy, whereas of the 8 patients surviving for periods of 1½ to 6 years, 7 had had some form of radiation therapy. He did, however, include cases showing periarteritis in his study and some of these may have been suffering from Wegener's granulomatosis. Glass[10] at the same symposium reported 4 cases treated by radiation to large fields in small doses of 50 to 80 r weekly. One patient had died of agranulocytosis, but 3 were alive from 7 months to 3 years after treatment. Subsequently, Benton[3] in a personal communication reports that these 3 patients were still alive 5 to 8 years later, although one had required subsequent treatment on several occasions. He mentions 4 other patients who did not respond as well to similar small doses. Howells[15] at the same symposium, reported upon 2 patients who were well for 6 months following small doses of radiation over a 7 week period, neither of whom had shown response to cortisone in large doses. Howell[14] described a case at the Royal Society of Medicine showing marked improvement and no active inflammatory process 3 months after 2200 r tumor dose had been given in 11 days at 220 KVP. This patient is subsequently understood to have developed a squamous cell carcinoma of the nasal septum and to have survived an extensive surgical procedure for at least 3 years.[16] Ellis[6] reported a most interesting case which had shown a very dramatic response to a total dosage of 300 r given in the course of one month. This patient was still well and showed no evidence of activity of her disease 10 years following the treatment,[8] although she still had a small fistula which had failed to respond to several attempts at surgical closure. The patients of Glass and Ellis appear to be the only ones described in the literature to have survived for any length of time, and it is noteworthy that all had been exposed to very low dosage of radiotherapy.

More recently, the tendency seems to be to combine the effects of steroids and radiation as described by Howells[15] and Hultberg and associates,[17] and as recommended by Ellis[7] and by Singh and co-workers.[26] It is too early to assess the merits of this combined therapy but it would appear that if there is evidence of systemic manifestations, x-ray therapy is inadequate to control the process.

PRESENT SERIES

In January, 1955, a young man was referred to the Department of Radiotherapy of The Johns Hopkins Hospital with the diagnosis of lethal mid-line granuloma. At that time no reports were to be found in the literature of the effect of low dosage radiation at what may be called anti-inflammatory levels. As this condition has the appearance of an acute inflammatory process, this method of treatment seemed worthy of trial and it was, therefore, adopted. Since then 4 other cases have been seen and 2 others found on retrospective survey of the hospital records. The histories of these 7 patients, treated in a variety of ways, will be briefly reviewed.

CASE REPORTS

Case 1.—O. M., a 60-year-old white man who had been a gas and oil distributor, was first seen in October, 1946. He gave a history of nasal congestion for 15 years which had been unrelieved by the usual measures. A submucous resection in March, 1946, had been performed without benefit,

and in June, 1946, antibiotics and sulfonamides had been given without effect. For three weeks an ulcer had been present on the palate, followed by progressive destruction of the left maxilla accompanied by high fever and foul-smelling discharge. In December, 1957, a radical excision of the maxilla was performed. Histologic reports were of nonspecific chronic granulation tissue. In February, 1948, fever was still present and during the course of 2 months the patient received radiation therapy in doses of 100 r at 200 KVP on 10 occasions. Marked local improvement occurred and the fever subsided. In August, 1948, the patient was readmitted with fever and further ulceration of the facial tissues on the right. Another extensive operation was performed and the patient died of meningitis soon thereafter. On review of the histologic sections, the diagnosis of lethal mid-line granuloma was made.

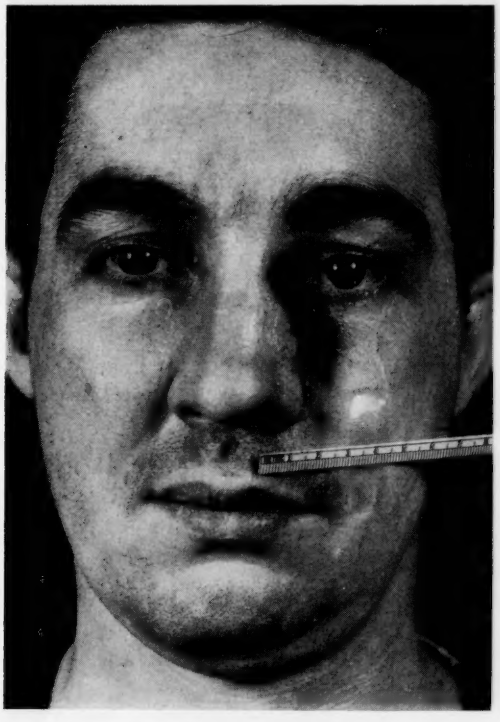

Fig. 1.—Case 3 after first excision (March 15, 1955). (By permission of the British Journal of Plastic Surgery.)

Case 2.—R. C., a 45-year-old white man who worked as a coal miner, was first seen at The Johns Hopkins Hospital in June, 1945. His symptoms were ulceration of the right nasolabial fold, loss of the side of the nose and upper lip, and a perforation of the septum and right upper gum. In 1927, his nose had been broken, and in 1941 a submucous resection had been performed. The patient suffered from chronic nasal discharge, and ulceration of the skin had been present for about 3 weeks. A biopsy was reported as showing the changes of "lymphosarcoma of mixed cell type." The patient was given a course of x-ray therapy consisting of 8 doses of 200 r each to a 4 by 5 cm. portal and 3 doses of 200 r through a 10 by 10 cm. portal, delivered to the right side of the face at 200 KV. The ulceration was arrested and 8 months later, in February, 1946, no active disease was found. The patient was advised to have a plastic repair of the destroyed area.

Multiple procedures were attempted with continued breakdown of the flap, but finally a new nose was constructed in 1953. In February, 1954, further breakdown of the nose occurred. The maxilla was removed and the right eye exenterated, the tissue on this occasion being reported as showing lethal mid-line granuloma. Review of the sections from 1945 disclosed that the original diagnosis of lymphosarcoma had been erroneous. The patient was subsequently placed on steroids and 12 months later no further activity was apparent.

Case 3.—F. H., a carpenter, 33 years of age, was first seen at Christmas, 1953. The patient noted that his left nostril was blocked. He was given penicillin with some temporary improvement. In January, 1954, biopsies taken from the nose and left antrum were normal. In March, 1954, the left side of the nose became blocked again and a node was discovered in the left side of the neck. Biopsies again were negative. In November, 1954, a left Caldwell-Luc operation resulted in the diagnosis of lethal mid-line granuloma. X-rays at this time showed destruction of the walls

Fig. 2.—Case 3, showing advanced disease on the right; disease controlled on the left (June 15, 1956). (By permission of the British Journal of Plastic Surgery.)

of the left antrum and of the ethmoid air cells. The patient was given small doses of cortisone and referred to the Radiotherapy Department of The Johns Hopkins Hospital. On examination there was a tender swelling at the left inner canthus and a large ulcer on the left side of the hard palate. There was a foul discharge from the left side of the nose, which was completely obstructed. It was elected to treat him with small doses of radiation, and on alternate days 100 r was delivered to the left side of the face through a 5 by 6 cm. portal at 250 KVP with great relief of fever and discharge, and with cleaning of the edge of the ulcerated area. The improvement, however, was temporary and in February the patient developed edema of the left eye with ulceration of the

skin at the left inner canthus. The left nasal and antral bones and the left side of the palate were excised (Fig. 1), but soon thereafter the right side of the palate and the nose became affected and began to ulcerate. In February, 1956, an extensive resection of the right maxilla and exenteration of the right orbit were performed. In March, the patient developed an ulcerated lesion of the left side of the larynx and pyriform sinus and 10 doses of 100 r each at 250 KV were delivered on alternate days to this area. This resulted in immediate relief of pain and clearing of the ulceration of the larynx. At the same time the destructive area in the right side of the face progressed, although again low dosage radiation served to clean the margins of the ulceration (Fig. 2). At this time the patient was receiving large doses of cortisone. In spite of this and in spite of further attempts at excision, the ulceration progressed to involve the whole of the right maxilla and palate until the pharynx was exposed. The patient died in October, 1956, without any return of the painful ulceration of the larynx and without any extension of the disease on the left side of the face, where the condition had apparently started.

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Fig 3.—Case 5, showing multiple skin nodules (March 12, 1958).

Case 4.—F. A., a white male salesman, 43 years of age, had had recurrent sore throats from 1948 onward. In 1951 a tonsillectomy was performed, but this did not prevent further sore throats. In October, 1954, the patient had a severe attack of upper respiratory infection and was admitted to another hospital, where he was hospitalized for 2 months with fever, nasal discharge, and crusting of the nose. Biopsies were nonspecific. In May, 1955 a furuncle in the right nasal vestibule eroded to the exterior and a large portion of the nasal cartilage was removed without pain. In August, 1955 the patient was seen at The Johns Hopkins Hospital with a large septal perforation. He was given antibiotics with some improvement. In September multiple skin nodules appeared

on the legs and abdomen, a biopsy of one revealing changes compatible with erythema nodosum. In October the patient developed a swelling over the left side of the nose. A biopsy from the septal perforation showed lethal mid-line granuloma. The patient was given large doses of steroids but the destructive process progressed and the skin lesions became much larger. He developed hoarseness with laryngeal ulceration. A skin lesion showed some improvement after a dose of 300 r but a similar dose to the larynx produced little change. He died on December 27, 1955, and the autopsy findings were reported in full by Spear and Walker.[29]

Case 5.—M. C., a 71-year-old white woman, gave a history of nasal obstruction for 18 years. When she was first seen in March, 1957, she had complete occlusion of the left nostril by a tumor mass. X-rays showed destruction of the right lateral antral wall and floor of the orbit. She was thought to have a carcinoma of the right antrum and a biopsy was reported as suggestive of squamous carcinoma. The patient was started on a course of radical x-ray therapy and between April 1 and June 27, 1957, a tumor dose of 6,500 r was given. The time interval was prolonged because an unsuccessful attempt was made to persuade her to have an operation on the antrum. The nasal obstruction cleared markedly but in October it recurred. A radium implant of the left nasal cavity was performed. In January, 1958, the patient developed an ulceration of the left pharyngeal wall with pain on swallowing and the next month a purpuric mass appeared on the left thigh, along with multiple other skin nodules (Fig. 3). Doses of 150 r on alternative days for 11 treatments produced no change in a large skin lesion on the left leg except that the pain disappeared. She developed respiratory symptoms and on April 11, 1958, she died. At autopsy, the skin, muscle, nose, sinuses, cervical nodes, and adrenals were all found to be involved in a process which histologically had the appearance of lethal mid-line granuloma. The original section was reviewed and the diagnosis of squamous cell carcinoma was found to be erroneous.

Case 6.—J. E., a 74-year-old white man, was first seen in September, 1956, complaining of irritation on the left side of the throat of 6 to 7 months' duration. On examination there was an ulcerative tumor of the left upper pole of the tonsil and adjacent soft palate. Biopsies of this area showed chronic inflammation. After one month of treatment with antibiotics had failed to produce a response, an excision of the area was undertaken. The tissue on this occasion showed the changes of lethal mid-line granuloma. The patient was referred to the X-ray Department and between Oct. 18 and Oct. 31, 1956, 7 doses of 150 r each were delivered through opposing 5 by 6 cm. portals at 250 KVP. There was immediate healing of the area and to date the patient has shown no recurrence of activity.

Case 7.—N. N., a white girl 21 years of age, came to the Hospital in September, 1956, when the right nostril became blocked. The patient was treated for sinusitis. In February, 1957, she developed a right infraorbital swelling and lacrimal abscess. She was found to have erosion of the anterior turbinates on the right with perforation of the septum and the palate. At that time the patient had a high fever. Biopsies showed only chronic inflammation, but in spite of antibiotics the destructive process extended posteriorly and to the left. At another institution, the patient was given a course of x-ray therapy to the right lateral antrum, a total skin dose of 3,000 r being delivered in 7 treatments through a 10 by 10 cm. portal at 200 KVP. At that same time, she received ACTH in the dosage of 30 units daily. She had some transient pain in the right lower quadrant of the abdomen. When the patient was first seen at The Johns Hopkins Hospital on April 10, 1957, there was ulceration of the palate as far as the alveolar margin on the right with extension posteriorly to the region of the soft palate and across the mid-line. The edges of the ulceration were clean and appeared to be healing; however, on April 18 a radical resection of the maxilla was performed, and a diagnosis of lethal mid-line granuloma was made. The patient did quite well for 2 months but the pain and swelling of the right side of the nose recurred and a biopsy showed further active disease. She was referred to the X-ray Department and between July 19 and July 31, 1957, she received 6 doses of 150 r each through a 6 by 8 cm. portal at 250 KVP. There was complete relief of the swelling and the operative cavity once more became quite clean and painless. She remained well until February, 1958, when she had a severe attack of acute abdominal pain, was admitted to the hospital, and at laparotomy was found to have necrosis of the right ovary with severe peritonitis. On February 27, she died. At autopsy there was involvement of the myocardium, the left adrenal and kidney, the abdominal nodes, and the retroperitoneal tissues with lethal mid-line granuloma. There was no evidence of recurrence of disease in the region of the face.

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DISCUSSION

A survey of the literature prior to 1955 reveals that no patient treated by antibiotics, extensive surgery, high doses of radiation, or by steroids had survived from the time of diagnosis of the disease for more than a few months. The only reported cases of survival for any reasonable length of time, in fact, are those in the papers by Glass[10] and Ellis.[6] An analysis of the cases treated in this Department demonstrates that control of the local lesion was obtained only by small dose radiation therapy (Table II). Case 3 demonstrates well the control of the areas which received small doses of radiation prior to extensive surgery and the rapidity of progression of the disease in areas which were treated solely by excisional surgery. Case 2 was treated by small doses of radiation followed by operation, and Case 6 was treated by radiation alone, although it must be admitted that the biopsy specimen from this patient was not entirely satisfactory and there is some slight doubt as to the diagnosis. Case 1 was treated by surgical intervention initially and control of the disease was obtained only when radiation therapy was given, this control lasting only a few months. High doses of radiation given in Cases 5 and 7 served to delay the progression of the disease but in neither case was the benefit of any duration. Case 4 received steroid therapy alone and at no time was the relentless extension of the disease halted.

Any discussion of the mode of action of radiation in improving the local situation and in leading to arrest of the spread of the disease can only be speculative, but it seems reasonable to postulate that, if this is either an inflammatory process or a hypersensitivity response, small dosage radiation, by analogy with comparable experience in other conditions, might be expected to be beneficial. The lack of control by radical or local surgery alone suggests that the trauma of the operation may lead to extension of the disease at the limits of the excised area. It is at least possible that high dosage of radiation might have a similar action and we do not agree with those who advocate tumoricidal doses.[4,28] It is postulated that small doses of deep x-ray therapy should be given as early as possible in the course of the disease, and that surgical excision should be delayed until the process has been controlled for at least several months. This policy may fail, but the only long-term survivors of this disease have been so treated, and the cases in the present series lend some support to this suggestion. Steroids should be given if the process is more extensive, or if there is evidence of systemic involvement. The greatest difficulty appears to be in the recognition of the disease early in its development, since the symptoms exhibited approximate those and whereas heretofore the diagnosis has usually been made by a process of exclusion after major surgical biposies, it is submitted that the diagnosis of lethal mid-line granuloma should be considered as soon as the patient is first seen and malignant disease has been excluded.

SUMMARY

1. The pertinent literature on lethal mid-line granuloma is briefly reviewed, and it is noted that the only cases in which relatively long-term healing was obtained were those in which small doses of radiation were given prior to operation.

2. Seven cases treated at The Johns Hopkins Hospital are reviewed. They were treated in different ways and the results lend support to the idea that low dose radiotherapy is the treatment of choice at present.

3. Extensive surgery and radical radiotherapy would seem to be contraindicated in this condition and serve only to enlarge the scope of the destructive process. Small dosage radiation should be given as early as possible in the course of the disease and not until a reasonable period of healing has elapsed should reconstructive surgery be attempted. Steroids should be reserved for later cases and for those showing systemic involvement.

4. A plea is made for earlier diagnosis, since it appears that healing is more likely to occur in the early stages of the disease.

TABLE II. RESULTS OF TREATMENT OF MID-LINE GRANULOMA CASES (THE JOHNS HOPKINS HOSPITAL)

	TREATMENT	CONTROL	RESULT
1.	Surgery + 1,200 r/60	−	Died 1 year
2.	2,200 r/11 + surgery + steroids	Partial	Alive 11 years
3.	Left, 500 r/10 + surgery	+	
	Right, surgery + steroids	−	Died 2 years
4.	Steroids	−	Died 4 months
5.	6,500 r/60 + radium	Partial	Died 1 year
6.	1,050 r/14	+	Alive 3 years
7.	3,000 r/7 + steroids + surgery	3 months	
	900 r/12	7 months	Died 1 year

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Cardiovascular

BLOOD PRESSURE LABILITY: A CORRELATIVE STUDY

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IT IS a commonplace observation that the blood pressure of some hypertensive subjects varies widely while remarkably consistent readings are obtained from others. This phenomenon was observed by the authors in a study of the relevance of psychological factors to blood pressure in essential and renal hypertensive populations[1]. The systolic and diastolic blood pressure of some untreated patients in that study varied no more than a few millimeters of mercury in serial weekly measurements whereas the blood pressure of others exhibited variations of as much as 40 or 50 mm. Moreover, the magnitude of blood pressure variation tended to remain relatively constant for a given patient during several months of observation. Inspection of the medical histories, physical examinations, laboratory and x-ray studies of these subjects failed to reveal correlates of the blood pressure variability. There was a suggestion that an inverse relationship between the height of the blood pressure and its variability existed. This suggested correlation is being studied in a larger population.

The most striking feature of the blood pressure variability in these populations was its association with certain aspects of the behavior and personality of the patients. In semidirective interviews the subjects whose pressure showed more variation seemed unusually alert and restless. They asked many questions about the course and prognosis of their disease, and complained of many minor bodily symptoms which were referable to the musculoskeletal, cardiovascular, respiratory, and gastrointestinal systems. They watched intently as the mercury column rose and fell during blood pressure measurements, and often, during clinic visits, squirmed in their chairs and tapped their feet. Their speech and manner showed a certain dramatic quality. Sometimes they gasped, spoke loudly, looked frightened, or burst into tears. Nevertheless, they were regular in attendance, stated that they liked the doctor, and could be readily reassurred

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temporarily. Complaints of minor symptoms in one bodily system were usually terminated after reassurance but were commonly "replaced" by complaints in another area. In summary, it was observed that those patients whose blood pressure varied widely exhibited behavior which could be characterized as hysterical, hypochondriacal, and anxious during their interaction with a clinic physician.

The group whose blood pressure showed little variation was generally quieter, less dramatic, and more stolid. They verbalized less concern about their disease, and seldom asked about prognosis. Almost never did they watch their blood pressure being measured. Bodily complaints were few, and were described briefly in a matter-of-fact way. Rarely did they seek reassurance from the examining physician. The hysterical, anxious, and hypochondriacal features which appeared to be prominent in the group with variable blood pressures were not observed in these patients.

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However, such qualitative and subjectively based descriptions of blood pressure variability on the one hand and personality and behavior on the other are of little value to others. Therefore, the authors turned first to the literature and then to an experiment which, it was hoped, would clarify these observations.

The factors which transiently influence blood pressure readings, in the absence of treatment, may be arbitrarily divided into three groups:

1. Methodological Factors.—The type of sphygmomanometer, width of cuff, rate of fall of the mercury column, snugness of fit of the cuff, eye level of the observer, and pressure with which the stethoscope is applied to the arm may introduce transient errors into blood pressure readings.[2,3]

2. "Host" Factors.—Among these may be included the effects of posture, of respiration, the "Traube-Hering" waves, and differences in readings from one arm to the other.[4,5]

3. "Enviromental" Factors.—It has been shown that the time of day, the person taking the blood pressure, the topic being discussed with the patient, the administration of placebos, and the day's events have transient effects of considerable magnitude.[6-10] Some of these environmental factors may be intimately linked with a host factor of personality.

Many of these factors can be controlled. The blood pressure can be measured carefully in the same arm, at the same time of day, with the patient in the same position, by the same observer using the same calibrated mercury sphygmomanometer. The readings can be made at the beginning of the visit, presumably before the patient is either alarmed or reassured by interaction with the examining physician. Blood pressure readings ought not to be made in the presence of hyperventilation, sighing, or other respiratory abnormalities. All patients should be untreated during the period of observation. With these factors controlled, variations in blood pressure that occur are somewhat more likely to be related to the personality of the patient and his perception of, and reaction to, his life events.

The hypothesis we developed by observing patients' behavior in an interview setting and serially measuring their blood pressure was: in hypertensive population there is a significant positive correlation between blood pressure variability and the personality features of hysteria, hypochondriasis, and anxiety.

Prior experience with serial interviews and blood pressure measurements in normotensive medical students led us to believe that the same correlation between blood pressure variability and personality is not unique for hypertensive subjects but can also be demonstrated in a normotensive group. The present study was designed to test this hypothesis.

METHODS

Seventeen patients with essential hypertension and 17 normotensives served as subjects. The criteria for diagnosing essential hypertension are described elsewhere.[1] There were 15 women and 2 men in each group. All subjects in both groups were southern-born Negroes between the ages of 20 and 49 who had resided in Chicago for from 2 to 28 years. Employment, marital status, previous experience with physicians and hospitals, and level of education were similar in the two groups. All subjects were obtained from the admitting clinic of the University of Illinois Research and Educational Hospitals, and were referred to us by a nurse unfamiliar with the purpose of the study. Ten hypertensive patients sought help for conditions unrelated to blood pressure and had no prior knowledge of their disease. The normotensive patients sought medical attention for such conditions as hemorrhoids, chronic cervicitis, Trichomonas vaginitis, uterine fibromas, low back pain of various origins, and infertility. Aside from the disorders which prompted them to seek treatment, no subject exhibited serious illness or cardiovascular disease other than essential hypertension. There were no subjects with diabetes mellitus, chronic urinary tract infections, gross obesity, or mental disease of psychotic proportions.

Blood pressure was measured in the left arm by the same observer while the subject was seated. Readings were made according to the recommendations of the American Heart Association.[2] Each subject was seen always in the same clinic room and all determinations were made between 9:30 and 11:00 A.M., 2 hours or more after the subject's usual breakfast. The blood pressure was measured after the subject had been seated alone for 10 minutes. At the end of that time the investigator entered, and took the blood pressure without saying anything except "good morning." The blood pressure readings made on the first three visits were not included in the data. We anticipated that at least that many visits would be required for the subjects to become accustomed to the setting and examiner and exhibit pressures more usual for them. Subjects were examined at intervals of one or 2 weeks. The periods of study varied from 4 to 12 months, and were usually terminated by pregnancy, or need for an operation, or pharmacotherapy. No drugs other than mild analgesics were taken by the subjects during this time. No diet changes were made; and in no subject did body weight vary more than 6 pounds. The number of blood pressure determinations made under these conditions ranged from 7 to 18 for each subject.

In a given population, unselected with respect to blood pressure levels, the blood pressure resembles a continuous variable and approaches a normal distribution.*[11-13] A simple mathematical expression of blood pressure variability

*In fact, the distribution of blood pressure exhibits some positive skewness; i.e., there are more readings and a wider range of readings above the mode than below it.

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was developed. Seven blood pressure measurements were randomly selected for each patient. The standard deviations of the 7 systolic and 7 diastolic readings were determined. Thus four lability categories were established: standard deviation of 7 systolic blood pressure readings and of 7 diastolic readings for each member of the hypertensive group and standard deviation of 7 systolic readings and of 7 diastolic readings for each person in the normotensive group. The size of these standard deviations was used as the basis for ranking the subjects by variability of systolic and diastolic blood pressure. The assumption was made that a large number of blood pressure readings on the same individual would exhibit a normal distribution. Plots of 11 to 18 blood pressure readings for 5 randomly selected patients supported this assumption.

TABLE I. BLOOD PRESSURE VARIABILITY

NORMOTENSIVE PATIENTS				HYPERTENSIVE PATIENTS			
		STANDARD DEVIATION				STANDARD DEVIATION	
PATIENT	RANGE	SYSTOLIC (MM. Hg)	DIASTOLIC (MM. Hg)	PATIENT	RANGE	SYSTOLIC (MM. Hg)	DIASTOLIC (MM. Hg)
1	116/62–130/88	3.75	4.20	1	160/ 95–230/132	12.62	4.53
2	116/68–140/88	3.91	2.82	2	170/104–212/126	7.17	3.58
3	102/62–130/80	4.00	2.07	3	150/ 92–180/112	4.93	3.65
4	126/72–140/88	2.46	2.42	4	120/ 92–180/110	6.00	3.02
5	106/62–140/76	4.43	2.56	5	144/ 94–168/104	4.38	1.32
6	108/70–130/72	3.00	1.15	6	150/ 98–176/116	5.10	2.82
7	114/60–138/70	3.83	1.36	7	148/102–182/108	5.63	2.22
8	112/70–132/80	2.83	2.00	8	170/118–218/130	9.03	2.92
9	120/82–134/82	2.02	1.50	9	188/116–234/132	6.10	2.12
10	116/68–146/84	5.43	2.56	10	210/116–240/138	4.90	3.33
11	96/60–122/66	3.78	1.00	11	190/108–210/128	4.81	2.77
12	118/64–140/88	4.11	3.47	12	156/108–176/112	3.20	1.36
13	122/80–144/84	3.62	0.93	13	172/126–186/136	2.10	1.28
14	92/60–130/78	6.30	2.93	14	188/108–230/112	5.57	1.10
15	120/72–138/80	3.11	1.38	15	162/104–176/106	2.44	1.01
16	88/58–120/78	5.06	3.40	16	172/100–190/106	2.61	0.90
17	128/76–142/88	2.40	1.52	17	168/112–176/118	1.72	0.88

To assay the relevant personality characteristics, we employed the Minnesota Multiphasic Personality Inventory (MMPI), an instrument that is independent of observer judgments. This test is a lengthy questionnaire, empirically derived from a study of a large population of mentally ill and healthy patients by the combined efforts of a psychologist and a physician.[14] It has the further advantage that it gives numerical scores from which assessments can be made of the personality trends in which we are particularly interested. On either the fifth, sixth, or seventh clinic visit, the patients were required to complete the MMPI.

In each of the two groups, the patients were ranked from 1 to 17 on the basis of the magnitude of the systolic standard deviations, from 1 to 17 on the basis

of the diastolic standard deviations and from 1 to 17 on the basis of the scores of the various scales of the MMPI. We preferred to make no assumptions about the distribution of the MMPI scale scores in our two populations and intended subsequently to employ partial correlations. Therefore, the nonparametric Kendall rank correlation was chosen to compare blood pressure lability with the psychological variables.[15]

RESULTS

The systolic and diastolic ranges and standard deviations for the normotensive and hypertensive subjects are shown in Table I. In the hypertensive group, the Kendall rank correlation coefficient, Tau, between ranks by systolic and ranks by diastolic lability was .67 ($P < .001$). Thus those hypertensive subjects whose systolic blood pressure varied the most also exhibited a greater variability of diastolic blood pressure. A similar relationship between systolic and diastolic lability was obtained in the normotensive group (Tau = .29, $P < .05$) except that the correlation was not nearly so high.

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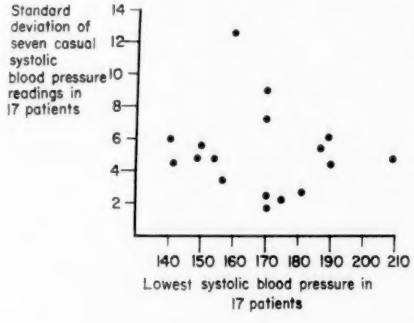

Fig. 1.

The possibility existed that a systematic error in the blood pressure determinations that increased as the readings became larger might introduce an increase in the standard deviations and a quantitatively greater error especially in the hypertensive group. To examine for this possibility, the systolic and diastolic standard deviations were plotted against the lowest systolic and diastolic blood pressure readings obtained for each of the 17 hypertensive subjects. It can be seen (Figs. 1 and 2) that the magnitude of the standard deviation showed no rise as the lowest blood pressure increased. Therefore, the variation in blood pressure readings, as reflected in the standard deviations was not influenced by a systematic error in the measurement that increased as the readings became larger.

The MMPI scale scores for both the normotensive and hypertensive groups did not differ significantly from those obtained in an earlier study of similar patient populations.[1] Those MMPI scales that correlated significantly with the blood pressure lability for both the hypertensive and normotensive groups are

shown in Table II. The Kendall rank correlation coefficients and the levels of significance are shown for both systolic and diastolic blood pressure in the normotensive and hypertensive groups. Three scales, the Hysteria (Hy), Hypochondriasis, K-corrected (HsK), and Psychopathic Deviate, K-corrected (PdK) were significantly and positively correlated with both systolic and diastolic lability in both the normotensive and hypertensive population. These scales assessed the degree to which personality characteristics found in hysterical, hypochondriacal, and impulsive acting out individuals were present in our subjects. The K-correction is a "suppressor variable" that "corrects" for the tendency of subjects to distort some of their answers to induce a more favorable impression of themselves.

Certain other scales correlated significantly and positively with one or more of the four lability categories. The Hypochondriasis (Hs) scale correlated with systolic and diastolic lability in the hypertensive group and with systolic lability in the normotensive group. The psychopathic Deviate (Pd) scale correlated with both systolic and diastolic lability in the hypertensive population. Five

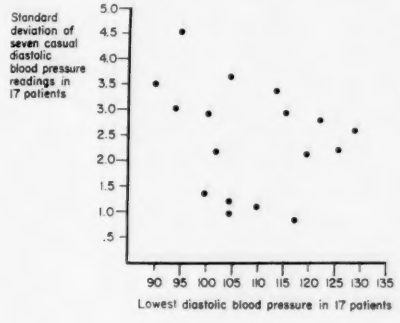

Fig. 2.

other scales, the Psychasthenia scales, uncorrected and K-corrected (Pt, PtK), Schizophrenia scale, K-corrected (ScK), Hypomania scale, K-corrected (MaK), and Paranoia (Pa) scale correlated with only one lability category. Eight MMPI scale scores did not correlate with any lability category. Those scales exhibiting no correlation were the Lie scale (L), the two validity scales (F,K), the Depression scale (D), the Masculinity Femininity scale (Mf), the Schizophrenia scale (Sc), the Hypomania scale (Ma), and the Taylor Manifest Anxiety (MaS) scale.

Table III shows the MMPI scale scores for the most labile and the most stable normotensive and hypertensive patients.

A brief description of the relevant scales follows. The Hy scale was empirically derived from a study of patients who had conversion hysteria symptoms. The scale has two components, one closely correlated with the Hs scale (to be described subsequently) and the other measured by a set of items which express a rejection of the possibility that the subject is neurotic.[14] A subject scoring high

on this scale could be described as immature, naive, needing social acceptance, suggestible, responsive to reassurance, narcissistic, and lacking in insight. The Hs and HsK scales are almost wholly made up of a set of physical complaint items characteristic of patients with a diagnosis of psychoneurosis, hypochondriasis. The actual syndrome is similar to the older term neurasthenia. A person scoring high on this scale could be described as concerned over bodily function and health, physically complaining, pessimistic, and dispirited. The Pd and PdK scales were derived chiefly from a study of a population generally considered to have a psychopathic personality. These persons are marked clinically by antisocial or asocial behavior that may take many forms. Such persons are impulsive, lacking in control of word or deed, irresponsible, tactless, talkative, adventurous, and frank. The Pa, ScK, MaK, Pt, and PtK scores were derived from a study of paranoid, schizophrenic, hypomanic, and psychasthenic patient groups, respectively.

When correlation is observed between two variables, there is always the possibility that this correlation is due to the association between each of the two variables and a third variable. It was stated earlier that the Hy, Hs, and HsK scales assessed in part the same personality variables. The correlation between the Hy scale ranks and the ranks in each lability category is generally higher than the other correlations. For these reasons, partial correlations statistically eliminating the effects of the Hy scale ranks on the correlations between lability and the other scales were carried out. When the Hy scale scores were held constant mathematically, none of the other scales, with a single exception (Pa scale), correlated significantly with any of the four lability categories. Therefore, although three scales correlated with all lability categories, there is probably a set of personality variables common to these three scales that determines this relationship.

TABLE II. SIGNIFICANT CORRELATIONS BETWEEN BLOOD PRESSURE LABILITY AND MMPI SCALE SCORES

	NORMOTENSIVE SUBJECTS			HYPERTENSIVE SUBJECTS		
	MMPI SCALE	TAU	LEVEL OF SIGNIFICANCE	MMPI SCALE	TAU	LEVEL OF SIGNIFICANCE
Systolic Lability	Hy	.43	$<.01$	Hy	.51	$<.01$
	Hs	.31	$<.05$	Hs	.34	$<.05$
	HsK	.39	$<.05$	HsK	.58	$<.001$
	PdK	.28	$<.05$	Pt	.45	$<.01$
	MaK	.29	$<.05$	PtK	.43	$<.01$
				Pd	.37	$<.05$
				PdK	.33	$<.05$
				ScK	.31	$<.05$
				Pa	.43	$<.01$
Diastolic Lability	Hy	.60	$<.001$	Hy	.63	$<.001$
	HsK	.56	$<.001$	Hs	.32	$<.05$
	PdK	.47	$<.01$	HsK	.44	$<.01$
				Pd	.38	$<.05$
				PdK	.50	$<.01$

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TABLE III. MMPI SCALE SCORES OF THE MOST LABILE AND THE MOST STABLE SUBJECTS

SCALE	HYPERTENSIVE GROUP		NORMOTENSIVE GROUP	
	MOST LABILE PATIENT	MOST STABLE PATIENT	MOST LABILE PATIENT	MOST STABLE PATIENT
L (Lie Scale)	46	46	53	50
F (Validity)	55	50	55	53
K (Validity)	64	53	72	48
Hs (Hypochondriasis)	84	44	60	52
HsK (Hypochondriasis, K-corrected)	95	44	72	52
D (Depression)	98	65	78	63
Hy (Hysteria)	98	47	80	52
Pd (Psychopathic Deviate)	89	50	68	61
PdK (Psychopathic Deviate, K-corrected)	95	53	79	57
Mf (Masculine-Feminine)	39	53	45	66
Pa (Paranoia)	76	44	65	65
Pt (Psychasthenia)	78	50	50	46
PtK (Psychasthenia, K-corrected)	99	52	69	43
Sc (Schizophrenia)	63	53	47	43
ScK (Schizophrenia, K-corrected)	78	57	64	40
Ma (Hypomania)	59	45	50	66
MaK (Hypomania, K-corrected)	63	45	55	65

An analysis of all items on the Hy, and Pd and Hs* scales was undertaken to attempt to delineate more clearly the personality characteristics differentiating subjects with labile blood pressure from the stable group. The 10 subjects whose blood pressure was most labile and the 10 with the most stable readings were identified in the combined normotensive and hypertensive populations. A determination was made of the number of patients in each group answering each item "true" or "false" on all items in the Hy, Pd, and Hs scales and the results tabulated in 2 times 2 contingency tables. A chi square test was applied to see if the labile group answered any item in a significantly different way from the stable population. Only 2 of a possible 112 items showed significant differences. Therefore the over-all scales rather than any specific cluster of items within the scales are related to blood pressure lability.

*The items on the Hs scale and Pd scale are identical with those on the HsK and PdK scales. The only differences are in the use in the latter scales of the suppressor variable, K.

DISCUSSION

The Hy scale assesses an aspect of personality that is closely correlated with magnitude of variability of blood pressure in both a normotensive and hypertensive group. This personality aspect is also reflected in the PdK and HsK scales. It may appear as part of a syndrome characterized as hysterical, hypochondrial, and unrestrained.

As outlined earlier, the HsK and PdK scales are correlated with blood pressure lability only because both these scale scores and lability are correlated with a third variable, the Hy scale scores. When the effects of the Hy scale were partialled out, the correlations between the HsK and PdK scales and lability were insignificant. Ten items used in scoring the Pd scale and 20 items employed in scoring the Hs scale are also utilized in scoring the Hy scale. Similarly, one item is common to the Pd and Hs scales. This overlap of items in the three scales may help explain the fact that all three are correlated with blood pressure lability but that mathematically only the Hy correlation with lability is important. In addition to the item overlap there is an overlap in the behavioral traits associated with the three scales. The Hy and Pd scales mirror tendencies toward emotional expressivity and action oriented (as opposed to thought oriented) behavior. The Hs and Hy scales indicate the degree to which the individual is aware of bodily symptoms and "uses" them to achieve emotional security. It was demonstrated that the entire Hy, HsK, and PdK scales rather than subgroups of items correlated with blood pressure lability. It is likely therefore that the overlap in behavioral traits in the three scales is a more important aspect of this correlation than the item overlap.

At the beginning of the study it was postulated that those subject whose blood pressure varied the most would exhibit higher scores on measure of hysterical, hypochondriacal, and anxious behavior. Part of this hypothesis was borne out, part was not. Anxiety as assayed by the Taylor Manifest Anxiety Scale and the Psychasthenia Scale was not significantly correlated with three of the four lability categories. On the other hand, the factor correlated with blood pressure lability in our patients was related to impulsive behavior, an association we had not foreseen.

Others have described relationships between personality variables and certain autonomically regulated body functions. Lacey[16] studied the frequency with which heart period and galvanic skin resistance varied in a healthy young adult population. He states, "There were some obvious personality differences between at least the extremes of our autonomic measures . . . . Such terms as manifestly anxious, hysterical, and overly reactive were suggested as characterizing a few subjects with the largest amounts of autonomic activity; at the other extreme we were tempted to apply such words as phlegmatic, repressed, resilient, lacking affect." Moreover, Lacey quotes Burch, Cohn, and Neuman[17] who have described spontaneous variations in resting peripheral blood flow and who observed similar personality differences. In the rare disorder, familial dysautonomia, the outstanding features of the disease are grossly defective autonomic regulation manifested by marked variations in pulse rate, blood pressure, sweating, body temperature, and gastrointestinal function, and by emotional lability of severe degree.[18,19]

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The personality variable delineated in our studies by the Hy, HsK, and PdK scales has also been described by other observers. It may be inversely related to a source trait described by Cattell.[20] This trait, called "ego strength" or "trait C," has a positive pole delineated by the following terms: "emotionally stable, free of neurotic symptoms, not hypochondriacal, realistic about life, unworried, steadfast, self-controlled, calm, patient, perservering and thorough, loyal, dependable." Those lacking in this trait have been described as "emotional, dissatisfied, showing a variety of neurotic symptoms, hypochondriacal, plaintive, evasive, immature, autistic, worrying, anxious, changeable, excitable, impatient, quitting, careless, undependable morally."

The role of genetic and enviromental factors in the determination of these autonomic and personality variables has not been widely studied. Cattell examined the inheritance of some aspects of personality and concluded that early life experience was a more important determinant of the "ego-strength trait" than genetic influences.[20] In a study of autonomic activity in twins, siblings, and unrelated children, Jost and Sontag[21] came to the conclusion that patterns of autonomic functioning may be at least partly determined by heredity.

Several authors have attempted to separate a labile group from the remainder of a hypertensive population. Perera demonstrated that hypertensive subjects with labile blood pressure have a better prognosis in terms of length of life and die of different causes than those whose blood pressure is relatively invariable.[22] Wolf,[23] Ferris,[24] and Stead in the discussion of their papers have all suggested in different words the same hypothesis: part of any arbitrarily defined hypertensive population may consist of patients who do not have the syndrome of essential hypertension but who instead exhibit a marked blood pressure lability and hysterical anxiety. They postulate that these subjects may show varying but elevated blood pressure readings in the physician's office, and, with no therapy other than a good patient-physician relationship, become normotensive in the clinic setting.

These studies and the present one may initiate the eventual characterization of subgroups of the larger hypertensive population. It may be possible to delineate members of a subgroup with labile blood pressure in this way. They exhibit certain neurotic behavior traits. They may live a relatively long life with hypertensive disease and are likely to die of a myocardial infarction or cerebral vascular accident.[22] A good patient-physician relationship with its opportunities for reassurance, for development of insight, and adaptation of newer, more favorable attitudes may mitigate the hypertensive disease. On the other hand, the subjects whose blood pressure is more unvarying do not appear to exhibit these neurotic behavior traits. Their life span is relatively short and they are likely to die of renal disease.[22] It is more difficult to see how the deliberate therapeutic use of the patient-physician relationship could benefit this group.

Formulation of the concept of a labile hypertensive group in whom psychological factors are intimately related to blood pressure implies a major presumption. This presumption is that the personality of the patient and his characteristic pattern of response to life events underlie the blood pressure variability. The commonly observed rise in blood pressure following situations perceived by

subjects as threatening, and the decrease in blood pressure after reassurance supports this view. However the statistical association we have demonstrated between some aspects of personality and lability of blood pressure can be explained in at least two other ways. Possibly, as Lacey[16] believes, truly spontaneous fluctuations in autonomic nervous system activity may serve as a stimulus of cerebral cortical mechanisms and induce certain behavior patterns. Finally, both blood pressure lability and personality may be related to a third factor, not currently identifiable.

SUMMARY AND CONCLUSIONS

Two homogeneous, populations, one normotensive and one hypertensive were studied. The standard deviations of systolic and diastolic blood pressures were determined and used as indices of blood pressure lability. All subjects in both populations were ranked by systolic and diastolic blood pressure lability and by scores on the scales of the Minnesota Multiphasic Personality Inventory (MMPI). There was a significant positive correlation in both populations between systolic and diastolic lability and scores on the Hysteria, Hypochondriasis (K-corrected), and Psychopathic Deviate (K-corrected) scales of the MMPI, i.e., the subjects whose blood pressure varied the most scored higher on measures of impulsive, hypochondriacal, and hysterical behavior.

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EPIDEMIOLOGIC STUDIES ON CARDIOVASCULAR-RENAL DISEASES: I. ANALYSIS OF MORTALITY BY AGE-RACE-SEX-OCCUPATION

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IN THE last decade, the epidemiologic method has been used to an increasing degree to investigate problems of the etiology of the so-called chronic degenerative diseases, e.g. cancer, arthritis, and cardiovascular-renal diseases. This methodology has made especially significant contributions—parallel with those of clinical-pathologic and animal-experimental work—to the elucidation of problems of the etiology of coronary heart disease. In particular, considerable data have been accumulated demonstrating differences in the occurrence of coronary heart disease among populations in different countries of the world. In addition, significant advances have been made in investigating factors accounting for these trends.[1-13]

Late in 1954, the senior author undertook to extend a basic program of research on the etiology of atherosclerotic and hypertensive diseases to encompass studies utilizing the epidemiologic method. It was hypothesized that the heterogeneous populations of the city of Chicago and the state of Illinois constituted excellent epidemiologic "laboratories". A long-term program of research was projected, beginning with an analysis of Chicago mortality data. The overall plan included eventual prevalence and incidence studies in strata of the living populations of Chicago and Illinois.[14,15]

The present report presents data on Chicago mortality by age, race,† sex, and occupation for the years 1951 and 1953.‡

This work was made possible by grant support from the Chicago Heart Association.

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**Special Research Fellow, National Institutes of Health, United States Public Health Service.

†Of the 283,730 nonwhites in the Chicago population, age 25-64 in 1950, 96.4% were Negroes,[16,17] hence the nonwhite death rates are essentially those for the Negro population.

‡At the onset of these studies, the 1953 data were analyzed because this was the earliest year for which census tract data were available on the Chicago mortality punchcards (see the second of these 3 papers, ref.[27]). Subsequently the 1951 data were also examined, thereby maximizing the reliability of the population base from the 1950 census.

TABLE I. CAUSES OF DEATH TABULATED AND THE CORRESPONDING INTERNATIONAL STATISTICAL CLASSIFICATION (ISC) LIST CODE (3-DIGIT).

CAUSE OF DEATH	ISC CODE #
Diabetes mellitus	260
Vascular lesions affecting central nervous system	330–334
Chronic rheumatic heart disease	410–416
Arteriosclerotic heart disease, including coronary disease	420
Other myocardial degeneration	422
Other diseases of the heart	430–434
Hypertension with heart disease	440–443
Hypertension without mention of heart	444–447
Nephritis and nephrosis	590–594
Diseases of the heart	410–443
Cardiovascular diseases	330–334, 400–468
Cardiovascular-renal diseases	330–334, 400–468, 590–594
All causes	001–999

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METHODS

The mortality data were obtained from the records of the Chicago Board of Health. Statistical tabulations were accomplished by the Board of Health's Division of Public Health Methods and its Tabulating Unit. The causes of death are designated according to the International Statistical Classification of Diseases, Injuries, and Causes of Death, Sixth Revision (hereinafter referred to as ISC).

The causes for which tabulations were made are listed in Table I. Diabetes mellitus was included because of the frequency of deaths due to cardiovascular-renal "complications" of this disease. The broad groupings—cardiovascular-renal (CVR) diseases, cardiovascular (CV) diseases and diseases of the heart —are included in order to facilitate comparison with earlier reports.[18-26]

Since the diseases under study have emerged as major public health and sociologic problems primarily because of the high morbidity and mortality toll among people in the productive middle decades of life, it was decided to concentrate upon the 25 to 64 year age group in the population.

In addition to the usual age, race, sex divisions an attempt was made to investigate the factor of occupation.* Occupational groupings were based on the occupation code from the death certificate—a coding similar to the major occupation classification used by the United States Bureau of the Census, 1950: 1, professional, technical, and kindred workers; 2, farmers and farm managers; 3, managers, officials, proprietors, except farm; 4, clerical and kindred workers; 5, sales workers; 6, craftsmen, foremen, and kindred workers; 7, operatives and kindred workers; 8, private household workers; 9, service workers, except private household; 10, farm laborers and foremen; 11, laborers, except farm and mine; 12, occupation not reported. For analysis, the occupations were first pooled into five groups and subsequently into two.[29] The latter divided the working population into essentially two broad categories, nonproduction "white collar" workers and manual-production or service "blue collar" workers.

*Subsequent reports present findings on death rates by age-sex-nationality-income-place of residence (urban-rural).[27,28]

In view of the fact that a significant per cent of CVR deaths are unattended by a physician, a specific analysis was made of the proportion of deaths certified by the coroner's physicians.

Population data used in the calculation of death rates were obtained from the published tables of the 1950 population census.[16,17] No attempt was made to correct for changes in population between the census year of 1950 and the years of study, 1951 and 1953. Since the mortality rates for the two years were similar, mainly the 1951 data are presented. All death rates are expressed as deaths per 100,000 population. Since the 1950 census reports contained the age, race, sex, occupation specific data for the Chicago Metropolitan Area only, a special tabulation was obtained from the Bureau of the Census giving these data for the city of Chicago.*

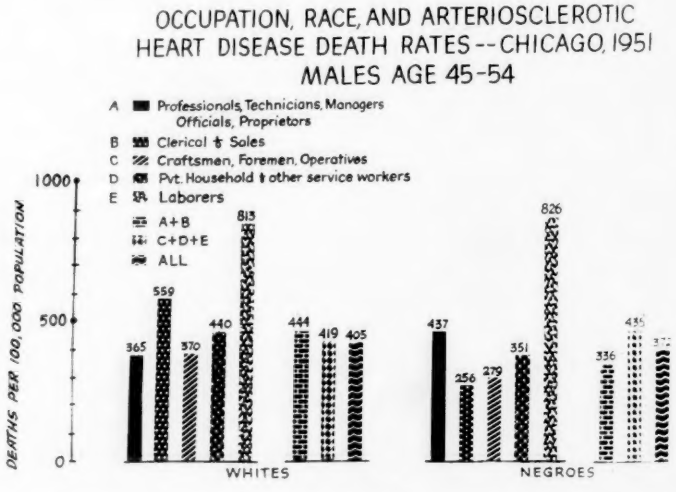

Fig. 1. C-V-R diseases are the totals for the broad grouping of the cardiovascular-renal (CVR) diseases; AS&DHD is arteriosclerotic and degenerative heart disease (ISC categories 420+422); ASHD is arteriosclerotic heart disease (category 420).

RESULTS

As is well known, the cardiovascular-renal diseases are as a group responsible for a high percentage of all deaths in middle-aged Americans (Table II and Fig. 1). This predominance of cardiovascular-renal mortality is largely the result of the high death rates due to diseases of the heart, particularly the entity arteriosclerotic heart disease (ISC 420) (Tables II, III, and Fig. 1). In terms of mortality, this entity—arteriosclerotic or coronary heart disease†—is clearly the number one public health problem for our middle-aged population today.

*It is a pleasure to acknowledge the cooperation and assistance of Mr. Howard G. Brunsman, Chief, Population and Housing Division, Bureau of the Census, in the accomplishment of this analysis.

†Since the ISC uses the term arteriosclerotic heart disease, this paper adheres to that designation, although it is recognized that atherosclerosis is the specific term describing the pathologic process in this disease.[45]

Arteriosclerotic Heart Disease (ASHD) (ISC 420).—This disease, like all others in the cardiovascular-renal group with the exception of rheumatic fever or rheumatic heart disease, increases markedly as a cause of death with age (Table III). In all four 10-year age groups in the 25 to 64 year span, the ASHD death rates are several times higher in males than in females. This sex differential declines with age, but is present to a significant degree in the 55 to 64 year age group. This differential is markedly less in nonwhites than in whites, principally because of the higher mortality rates in the nonwhite females.

Nonwhite male death rates for ASHD approximate those for white males, whereas nonwhite female death rates are consistently higher than those for white females. Hence, as already indicated, the sex differential in mortality for nonwhites is considerably less than for whites.

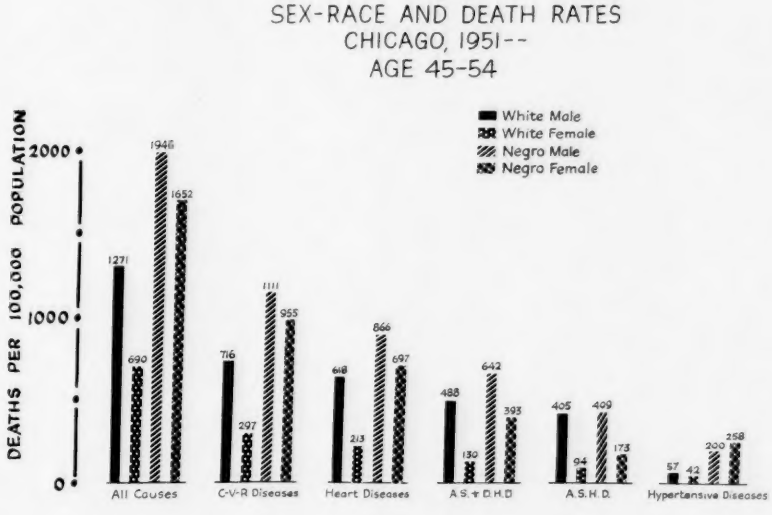

Fig. 2.

The data on occupation* reveal several apparent differences in mortality rates among the five groups of the employed population (Table IV and Fig. 2). Rates for both ASHD and all causes for males are apparently the highest in occupation group E (see discussion below). For white males, rates for groups A and C appear to be uniformly lower. For nonwhite males this pattern is not as consistent; the rates are lower for A, B, or C.

As a result of further pooling into two broad occupation groups—nonproduction white collar workers (A+B) and manual-production or service blue collar

*Because of the few deaths in the younger age group, the data on occupation are given only for ages 45 to 54 and 55 to 64. The death rates for the various occupation groups are based on deaths by place of occurrence rather than place of residence. However, this introduces an insignificant variation, since age-specific rates for white males for the various causes based on place of occurrence are very nearly equal to those by place of residence. The largest differences, and those are slight, appear in the infectious disease and accident categories.

TABLE II. AGE-RACE-SEX SPECIFIC DEATH RATES FOR ARTERIOSCLEROTIC HEART DISEASE AND FOR SEVERAL SUMMARY GROUPS OF CAUSES FOR CHICAGO RESIDENTS, 1951.

CAUSE	RACE	AGE AND SEX							
		25—34		35—44		45—54		55—64	
		M	F	M	F	M	F	M	F
Arteriosclerotic	W	8.3%*	3.0%	22.2%	6.2%	31.9%	13.6%	35.2%	20.8%
Heart Disease	NW	2.7	1.8	13.9	4.9	21.0	10.5	21.8	14.6
Diseases of the	W	20.2	13.1	37.5	21.4	48.6	30.9	52.0	40.2
Heart	NW	17.0	15.1	32.7	32.5	44.5	42.2	45.6	47.0
Cardiovascular	W	24.4	15.2	42.4	29.0	54.5	41.7	59.5	52.3
Disease	NW	20.5	23.1	41.1	44.8	55.4	55.4	57.5	65.4
Cardiovascular-	W	28.0	19.2	44.1	30.8	56.3	43.0	61.0	54.5
Renal Disease	NW	22.8	27.0	43.3	46.5	57.1	57.8	59.2	68.7
All Causes	W	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
	NW	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
Arteriosclerotic	W	14†	3	107	17	405	94	1051	340
Heart Disease	NW	13	6	123	36	409	173	929	446
Diseases of the	W	34	13	181	59	618	213	1554	657
Heart	NW	83	51	289	239	866	697	1945	1435
Cardiovascular	W	41	15	205	80	693	288	1779	854
Disease	NW	100	78	363	329	1078	916	2450	1996
Cardiovascular-	W	47	19	213	85	716	297	1822	891
Renal Disease	NW	111	91	382	342	1111	955	2525	2098
All Causes	W	168	99	483	276	1271	690	2989	1634
	NW	487	337	883	735	1946	1652	4261	3054

*Death rates expressed as percent of all causes.
†Deaths per 100,000 population.

workers (skilled, semi-skilled, and unskilled) (C+D+E)—the white male death rates for these two groups are very similar. The former group (A+B) has slightly lower rates for all causes and slightly higher rates for ASHD. Of the two occupation groups for nonwhite males, the nonproduction white collar workers (A+B) exhibit lower rates for both all causes and ASHD. Comparing whites and nonwhites, rates for all causes are consistently higher in the latter. ASHD rates are higher for whites than for nonwhites of the (A+B) group, whereas they are similar for whites and nonwhites in the (C+D+E) groups.

Occupation analysis for the white females reveals a pattern similar to that for the males, i.e. the highest rates for both all causes and ASHD are exhibited by Group E. The feature of equalization of rates with pooling observed for the white males is also repeated for the white females. The numbers involved in the detailed breakdown for the nonwhite females are of such small magnitude as to render them of little value for the purposes of this study. An additional point emerges, viz. the grossly higher rates for the "all other" group, which for the females is composed largely of housewives. This observation is apparent for both white and nonwhite females (see discussion below).

Hypertensive Disease (ISC 440-447).—All four sex-color groups demonstrate an increase in hypertensive disease death rates with age (Table III).

For the whites, death rates for hypertensive disease (440 to 447) and hypertensive heart disease (440-443) tend to be slightly higher in males than females. The ratio of male to female deaths from these causes, ranging from 1.0 to 2.0, is small compared to this ratio for ASHD. For the nonwhites, a tendency is noted for the females to have somewhat higher hypertensive disease death rates than the males, the ratio of female to male death rates ranging from 1.1 to 1.7 for the overall category 440-447.

A gross difference in death rates is noted between whites and nonwhites; the latter consistently exhibit several fold higher death rates due to hypertensive disease. The ratios of nonwhite to white death rates range from a minimum of 2.6 to more than 10.0. This differential is consistently greater for females than for males.

Other Cardiovascular-Renal Causes of Death.—

TABLE III. AGE-RACE-SEX SPECIFIC DEATH RATES* FOR SEVERAL CARDIOVASCULAR-RENAL DISEASES FOR CHICAGO RESIDENTS, 1951.

CAUSE	RACE	AGE-SEX							
		25–34		35–44		45–54		55–64	
		M	F	M	F	M	F	M	F
330–334	W	5	2	18	14	62	67	184	162
	NW	13	9	35	58	145	190	441	491
410–416	W	10	6	21	16	35	34	35	32
	NW	13	4	15	13	12	16	17	6
420	W	14	3	107	17	405	94	1051	340
	NW	13	6	123	36	409	173	929	446
422	W	6	2	30	13	83	36	227	120
	NW	38	24	86	81	233	219	528	453
420+422	W	20	4	137	30	488	130	1278	460
	NW	51	29	210	116	642	393	1457	899
430–434	W	4	2	10	5	38	11	90	26
	NW	4	7	15	20	61	46	110	51
440–443	W	1	1	12	7	52	38	142	133
	NW	15	9	47	89	148	236	348	478
444–447	W	1	0	4	2	6	4	10	12
	NW	2	9	22	25	52	25	41	26
440–447	W	2	1	15	8	57	42	152	145
	NW	17	18	69	114	200	258	389	504
590–594	W	7	3	9	6	23	9	43	38
	NW	11	13	20	13	33	39	76	102
260	W	2	0	4	5	9	19	39	76
	NW	2	4	0	5	12	23	41	57

*Deaths per 100,000 population.

Other myocardial degeneration (ISC 422): As previously indicated, the rates for this cause of death also increase with age (Table III).

For the whites, males have higher death rates than females, the ratio of male to female rates ranging from 1.9 to 3.0. For the nonwhites, the ratio of male to female rates is considerably smaller, ranging from 1.1 to 1.6. The nonwhites exhibit markedly higher death rates than the whites from this cause, the ratios

TABLE IV. AGE SPECIFIC DEATH RATES* FOR ASHD AND ALL CAUSES AND TOTAL NUMBERS OF INDIVIDUALS FOR SEVERAL OCCUPATION GROUPS OF MALES AND FEMALES CHICAGO 1951.

		Occupation Groups† White Males								
	AGE	A	B	C	D	E	A+B	C+D+E	ALL OTHER	TOTAL
420	45—54	365	559	370	440	813	444	419	153	405
	55—64	1123	1277	889	1168	1622	1189	1026	267	958
All causes	45—54	929	1453	973	1482	2641	1142	1197	644	1174
	55—64	2548	3017	2584	3340	5256	2748	3031	906	2613
Population	45—54	47135	32357	82192	17270	9353	79492	108815	18958	207265
	55—64	30809	22938	65243	18412	10541	53747	94196	27367	175310
		WHITE FEMALES								
420	45—54	85	25	54	65	374‡	42	63	119	92
	55—64	162	193	146	222	250‡	183	182	385	330
All causes	45—54	363	368	420	455	935	389	440	821	662
	55—64	1233	1133	817	889	1750	1167	952	1758	1570
Population	45—54	14036	35849	20490	10778	535	49885	31803	129912	211600
	55—64	8027	15529	12363	10350	400	23556	23113	126351	173020
		NONWHITE MALES								
420	45—54	437	256	279	351	826	336	435	199	372
	55—64	626	879	301	828	2251	763	969	379	772
All causes	45—54	1366	1324	1439	1537	4298	1342	2168	596	1728
	55—64	3754	3163	2355	3727	9809	3434	4648	1176	3489
Population	45—54	1830	2342	9662	6830	5328	4172	21820	7043	33035
	55—64	959	1138	3652	4105	2355	2097	10112	5016	17225
		NONWHITE FEMALES								
420	45—54	0	0	156	137	0	0	139	208	173
	55—64	712‡	837‡	186‡	160‡	0	769‡	163	514	440
All causes	45—54	611	772	1042	1009	2439	695	1079	1953	1593
	55—64	3203	2092	1395	1925	1724	2692	1764	3362	2965
Population	45—54	819	907	3839	5850	410	1726	10099	18745	30570
	55—64	281	239	1075	2494	116	520	3685	11480	15685

*Deaths per 100,000 population.
†A—Professional and semi-professional workers, proprietors, managers and officials.
B—Clerical, sales, and kindred workers.
C—Craftsmen, foremen and operatives.
D—Service workers.
E—Laborers.
‡Rate based on less than 5 deaths.

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ranging from 2.3 to more than 10.0. As a result of these trends of mortality, the ratios—deaths from ISC 422/deaths from ISC 420+422 and deaths from ISC 422/deaths from ISC 410-443—are higher for the nonwhites than for the whites (Table V). A high percent of ISC 422 deaths are certified by coroner's physicians (Table VI) (see discussion below).

When the deaths assigned to 420 and 422 are summated, the nonwhite death rates become uniformly greater than the white, the differential being of higher order for females than for males (Table III). The sex differential previously noted for ASHD *per se* remains of high order for the whites, and becomes of lower order for the nonwhites. The problem of ISC category 422 and its relation to coronary heart disease is discussed below.

Other heart diseases (ISC 430-434): Deaths coded to this category constitute only a small per cent of all heart disease deaths (Tables II and III). The death rates for this category tend to be higher for nonwhites than for whites. The white male rates consistently exceed the white female rates, whereas the nonwhite females have higher rates for the first two age intervals and lower rates for age 45 to 64. A high percent of ISC 430-434 deaths are certified by coroner's physicians (Table VI).

Vascular lesions affecting the central nervous system (ISC 330-334): These death rates increase with age. The death rates for white males and white females are approximately equal in all four age intervals, whereas the death rates for nonwhite females slightly exceed those of the nonwhite males in all but the first age group (Table III). The nonwhite rates are grossly higher than those for whites, the differential ranging from 1.9 to 4.5.

Nephritis and nephrosis (ISC 590-594): Relatively few deaths are involved in this category. However, it appears even from this limited information that white male and white female death rates do not differ appreciably, and that nonwhite rates are considerably in excess of white rates.

Chronic rheumatic heart disease (ISC 410-416): As previously mentioned, of the disease categories studied here this is the only one exhibiting no marked increase in rate with age. Though the rates are small, they appear to show a preponderance of deaths in the whites with little evidence of a sex differential.

Diabetes mellitus: Only in the two older age groups were there significant numbers of deaths. This is one of the few diseases having higher mortality rates for women than men, both white and nonwhite.

DISCUSSION

Several distinctive observations emerge from the data on cardiovascular-renal mortality rates in 25 to 64 year old Chicagoans. First, certain contrasts are evident in the sex-race data for coronary disease and hypertensive disease. A sex differential is predominant for the former, a race differential for the latter. These findings are fully in accord with numerous reports on morbidity and mortality from these diseases in the United States.[1-3,9-13,18-26,30-44] They may be safely regarded as established facts in the epidemiology of these two diseases in this country.

This contrast in the epidemiologic patterns of coronary and hypertensive disease strongly supports the thesis that these represent two diseases of different etiology and pathogenesis. This concept is corroborated by extensive data from other sources.[1-3,41-47]

Considerable evidence exists indicating that estrogenic secretion by the ovaries in premenopausal women is a key factor responsible for the sex differential in ASHD mortality.[3,45-49]

Much less is known concerning the basis for the far higher death rates in Negroes than whites due to hypertensive disease. Several hypotheses may be suggested. Negroes may be victimized more by infections and other pathologic processes in the genito-urinary tract—processes which may lead to hypertensive disease.[41-47] Negroes may have patterns of diet in early life and adulthood which may increase susceptibility to hypertensive disease. The patterns of discrimination and segregation which Negroes experience in the United States may induce psychologic stresses, strains, frustrations, etc. These primary central nervous system effects may be responsible for the greater occurrence of hypertension in this racial group.[41-47] Undoubtedly the race differential in hypertensive disease affords an important lead to etiology. In view of the complex of unsolved problems concerning basic mechanisms of this disease, the follow-up of this lead certainly seems to be highly worthwhile.

TABLE V. AGE-RACE-SEX SPECIFIC RATIOS OF OTHER MYOCARDIAL DEGENERATION (I.S.C. 422) DEATHS TO THE SUM OF ASHD (I. S. C. 420) AND OTHER MYOCARDIAL DEGENERATION DEATHS, AND TO DISEASES OF THE HEART (I. S. C. 410-443) DEATHS.

RATIO	SEX-RACE	AGE			
		25–34	35–44	45–54	55–64
422 / 420 + 422	M— W	.31	.22	.17	.18
	M—NW	.72	.41	.38	.38
	F— W	.60	.45	.23	.25
	F—NW	.84	.70	.57	.50
422 / 410 − 443	M— W	.18	.17	.13	.15
	M—NW	.46	.30	.27	.27
	F— W	.15	.22	.17	.18
	F—NW	.47	.34	.31	.32

With respect to arteriosclerotic heart disease, several other findings merit discussion, in addition to the aforementioned sex differential. The males of the two racial groups exhibited similar death rates. Thus, it would appear that coronary heart disease is a frequent cause of death among Negro males in Chicago. This finding is in accord with some data from other sources. However, several reports of clinical and pathologic studies suggest that coronary disease is relatively rare in American Negro males.[1-3,18-26,30,35,38,39,50-63] Further work is essential to clarify this problem. This is particularly in order, in view of the apparently paradoxical nature of some of the findings. On the one hand, hypertensive disease has a severalfold greater prevalence in Negro than white males; on the other

hand, the occurrence of coronary disease is no greater, and may even be less, in Negro than in white men. But it has been repeatedly shown experimentally and clinically that hypertension increases the risk of coronary disease.[1-3,9,14-15,41-45] Why, then, don't Negro men, with so much more hypertension, have more coronary disease? This fascinating and important problem of the coexistence and interdigitation of these two diseases in various age, sex, race, socioeconomic groups certainly merits further investigation in various parts of the country.

The data of this study further indicate a high Negro female death rate for ASHD, compared with white females. Therefore, the male: female ratio is considerably lower for Negroes than for whites. Here again, additional work is needed to validate this finding. If this be in fact a valid observation, it may be related to two phenomena in Negro women—i.e. frequent obesity and hypertension.[3]

With respect to ASHD death rate differences among the five occupation groups, their validity may be open to question. The usual procedure for designating occupation on death certificates may introduce significant discrepancies

TABLE VI. TOTAL NUMBER OF ALL DEATH CERTIFICATES AND PER CENT OF CERTIFICATES SIGNED BY CORONER'S PHYSICIANS FOR SEVERAL CVR CAUSES FOR THE AGES 45-54.—CHICAGO RESIDENTS, 1951.

CAUSE	W—M		NW—M		W—F		NW—F	
	NO. OF DEATHS	% COR.	NO. OF DEATHS	% COR.	NO. OF DEATHS	% COR.	NO. OF DEATHS	% COR.
330–334	128	6	48	8	142	7	58	12
420	840	35	135	67	198	25	53	42
422	171	71	77	69	77	47	67	79
430–434	79	76	20	50	23	52	14	57
440–447	119	7	66	3	89	3	79	3
410–443	1280	38	286	5	451	2	213	40
All causes	2635	29	643	35	1460	13	505	23

in terms of the census classification.[29,82-84] Thus, it is quite likely that many women with long periods of adult employment are classified as housewives at death. This likelihood is especially marked when death is due to chronic disease, including cardiovascular-renal disease, which is often preceded by lengthy morbidity, making employment impossible. This may well account for the apparently higher death rates for housewives, compared with working women. Further, for the males, the low rates for Group C (craftsmen, foremen, and operatives) and the high rates for Group E (laborers) may be factitious—resultants of a tendency to record skilled and semi-skilled workers as laborers on the death certificates.

In any case, pooling into two broad occupation groups yields age-specific ASHD rates that are very similar. This is true for both races. Thus, the Negro males of pooled group C+D+E have ASHD death rates approximately the same as those for white males of pooled groups A+B and C+D+E. This finding is in accord with the findings of several recent living population studies, at least with respect to white males.[1-3,9,14,15,64,80,81] It is now clear—contrary to a once widely

held impression—that coronary disease has high occurrence rates (both incidence and mortality rates) throughout all socioeconomic groups of the white middle-aged male population of the United States. In this country it is not a disease afflicting the executive, professional, managerial, entrepreneurial upper-class groups predominantly.

This observation poses a significant theoretic problem in relation to data from other countries on coronary heart disease. Considerable evidence is available indicating that in Japan, Italy, and Spain—and in the economically less developed countries of Asia, Africa, and Latin America—coronary disease is relatively rare in males of the lower socioeconomic strata.[1-3,10-13] As discussed in previous extensive reviews,[1-3] there are good reasons to conclude that this difference is indeed a valid one. In contrast to the findings from these international investigations, the data of this study suggest that death due to ASHD may occur as frequently in Chicago Negro and white laborers (on the lower rungs of the U.S. socioeconomic ladder) as in the white professional, executive, managerial group.

The question may, therefore, be asked: Does this apparent contrast pose an important challenge particularly to current concepts that habitual dietary habits account in a key way for epidemiologic patterns of coronary disease? Is this dietary-metabolic theory inconsistent with these epidemiologic findings? In the United States—in contrast to the bulk of the populations of the aforementioned countries—the lower socioeconomic classes consume diets similar to those of the upper strata, particularly in their content of total calories, total fats, saturated fats, cholesterol, and refined carbohydrates.[1-3,65-72] Thus virtually all U.S. population groups consume a diet that presumably is potentially atherogenic. In addition, all groups have high prevalence rates of obesity, hypercholesterolemia, and hypertension—abnormalities making for high risk of coronary heart disease in middle-age.[1-3,9,14,15] Hence the observed widespread atherosclerotic disease may certainly be interpreted as entirely consistent with the nutritional-metabolic theory.

Analysis of data on mortality due to the cardiovascular-renal diseases—and particularly hypertensive disease and coronary disease—is incomplete without attention to the categories, Other Myocardial Degeneration (ISC 422), Other Heart Diseases (ISC 430-434), and Vascular Lesions of the Central Nervous System (ISC 330-334). This is particularly true, since none of these three is a clearcut etiologic category, as is immediately apparent upon perusal of the specific listings. Thus, category 422—including such designations as *cardiosclerosis, cardiovascular sclerosis, myocardial sclerosis, myocardial degeneration with arteriosclerosis, atheroma of heart, or myocardium*—could conceivably encompass a significant number of coronary disease (ASHD) cases. Experience in a living population prospective study has been fully in line with this impression.[15]

This possibility is heightened by the inclusion in the ISC 422 category of *chronic myocarditis*—an anachronistic term once used to designate what is now recognized to be coronary heart disease.[73] This term is gradually falling into disuse, but is still employed to a limited degree.[74*]

*It is recognized that the designation, *chronic myocarditis*, is validly utilized for those rare cases of myocardial disease in which the pathology is truly the result of a chronic inflammatory process.

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Similarly, ISC 430-434 (Other Diseases of the Heart) includes *heart block, Stokes-Adams syndrome, auricular fibrillation, congestive heart failure, left ventricular failure, acute pulmonary edema due to heart disease or failure, organic heart disease, et al.* Patently, many cases of coronary and hypertensive disease might possibly be concealed in this category. It is not inaccurate to suggest that both ISC 422 and 430-434 are—from the standpoint of etiologic classification of heart disease—heterogeneous nondescript "waste-basket" conglomerations.

The category ISC 330-334 (vascular lesions affecting the central nervous system) encompasses *subarachnoid and cerebral hemorrhage*, the latter *with or without mention of arterial hypertension or arteriosclerosis; cerebral embolism and thrombosis; spasm of cerebral arteries; atheroma of cerebral vessels; cerebral arteriosclerosis; hypertensive encephalopathy; stroke.* The rules for coding provide that a case of hypertension or arteriosclerosis dying of a stroke be classified in the 330-334 group.* Hence—in relation to both atherosclerotic and hypertensive diseases—this category must also be evaluated. Based on the foregoing general considerations and the specific sex-race pattern of death rates for ISC 330-334 for middle-aged persons, it may be suggested that deaths in middle-age assigned to this category are made up largely of cases of hypertensive etiology.[75]

Similarly, the sex differentials in rates for ISC 422 and ISC 430-434 suggest that these listings consist—at least for whites—predominately of cases of coronary disease (occurring in normotensives or hypertensives).† In view of the numbers of cases involved, which are particularly substantial for ISC 422, this inference is a highly important one, requiring further validation.[74]

With respect to the different distributions of deaths between the races for these categories, particularly the preponderance of Negro deaths attributed to ISC 422, the problem of certification by the coroner's physicians requires evaluation. Thus for the age group 45 to 54, 47 to 79 per cent of the deaths assigned to ISC 422 and ISC 430-434 are certified by the coroner's physicians (Table VI). This fact—taken together with the finding that for ASHD itself 25 to 35 per cent (whites) and 42 to 67 per cent (nonwhites) of fatalities are certified by coroner's physicians—makes it highly important for heart disease epidemiologic research to pursue the special problem of sudden and unattended deaths.[39,74]

The finding of lower rheumatic heart disease death rates in middle-aged Negroes compared with whites is noteworthy, particularly since an opposite pattern was recorded for the years 1939-1941 for children and adolescents.[76-78] These observations are not necessarily contradictory. A significant percentage of Chicago's middle-aged Negro population in 1950 was reared in the rural South. Those age 45 to 54, for example, were children and adolescents (age 5 to 14) in the first and second decade of this century. At that time, U. S. northern cities had small Negro populations compared with today, and a much higher percent

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*Similarly, hypertensives dying of coronary thrombosis and infarction are classified in ISC 420. Obviously these rules result in under enumeration of hypertensive disease, particularly when only single cause coding is employed, as is generally the case.[74]

†As suggested in the previous footnote, the structure of the ISC, together with the rules for coding and the practice of single-cause tabulation, render impossible any routine statistical distinction among uncomplicated coronary disease, coronary disease n hyperitensives and coronary disease in diabetics. This problem is examined in greater detail in a later paper.[74]

of the total U.S. Negro population lived in the rural South. In 1915—in contrast to 1939 to 41 and thereafter—U.S. rheumatic fever and rheumatic heart disease death rates tended to be lower for Negro children and adolescents than for white.[79] This probably reflected a lower incidence rate of streptococcal infection and rheumatic fever in the predominantly Southern rural Negro population.

This phenomenon would tend to result 40 years later in lower Negro death rates due to chronic rheumatic heart disease in middle-age—as observed in the Chicago data. These possible patterns require further study. They are consistent with the concept that environmental factors—of the macro- and particularly the microclimate—are of importance in the etiology of rheumatic fever and rheumatic heart disease.[76]

It is reasonable to conclude that epidemiologic studies of the U.S. vital statistics on the cardiovascular-renal diseases yield data of value to investigators concerned with problems of etiology. It is also apparent that additional work is needed to evaluate the special problems in these vital statistics data. Finally, the inherent questions of research in this field compel development of epidemiologic studies in the living population.

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SUMMARY

An epidemiologic analysis was accomplished of cardiovascular-renal mortality by age, race, sex, and occupation for the Chicago population aged 25 to 64 in 1951 and 1953. The following were the principal findings: 1. Arteriosclerotic heart disease (ASHD) death rates were severalfold higher in men than women. This sex differential was greater in whites than nonwhites, chiefly because of the higher mortality rates in nonwhite females. Nonwhite male death rates for ASHD were similar to those for white males. No significant occupation group differences in age-specific ASHD death rates were observed for middle-aged men. 2. Hypertensive disease death rates were severalfold higher in nonwhites than whites, without a gross sex differential. This pattern also prevailed for cerebrovascular diseases, nephritis and nephrosis.

An evaluation was made of deaths attributed to Other Myocardial Degeneration and Other Diseases of the Heart, particularly in relationship to mortality patterns for ASHD and hypertensive disease. An analysis was also accomplished of deaths certified by the coroner's physicians.

Consideration was given to the bearing of the findings upon current theories concerning the etiology of these diseases.

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EPIDEMIOLOGIC STUDIES ON CARDIOVASCULAR-RENAL DISEASES: II. ANALYSIS OF MORTALITY BY AGE-RACE-SEX-PLACE OF RESIDENCE, INCLUDING URBAN-RURAL COMPARISONS

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THE PREVIOUS paper of this series presented an epidemiologic analysis of deaths due to the major cardiovascular-renal diseases in the Chicago population subdivided into specific age-race-sex and age-race-sex-occupation groups.[1] The present report is an extension of this analysis to include deaths by age-race-sex and place of residence for the populations of the city of Chicago and the state of Illinois. This encompasses an analysis based on income differentiation within Chicago and an urban-rural differentiation within the state.

METHODS

The Bureau of the Census publications for 1950 present data on median income for each of the 935 census tracts in the city of Chicago.[2] At the time this study was undertaken, the Chicago Board of Health had coded place of residence by census tract for all deaths occurring within the city for the year 1953 and thereafter. This enabled an analysis of cause-specific death rates for the year 1953 for the Chicago population by age-race-sex-place of residence. For this purpose the totality of census tracts was divided into five groups based on median family income in 1949—less than $2,000, $2,000-2,999, $3,000-3,999, $4,000-4,999, $5,000 and greater. The 1950 census data constituted the population base (the denominator in the death rate calculation.

The division of the population of the state of Illinois into three groups for study was dictated largely by the geographic code employed in 1951 by the Bureau of Statistics of the state of Illinois. All 1951 deaths† were classified ac-

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†The years 1951 and 1953 were the years of study in the previous paper.[1]

cording to this code. In detail this code provided for a rural-farm and rural nonfarm classification based on population density. This did not correspond to the 1950 national census classification, which was based on farm residence.

It was possible, however, with the 1951 geographic code to select the deaths of residents of incorporated municipalities having a population of 2,500 or more. The corresponding population figures (1950) for these areas were taken from the census reports which also listed data based on the 1940 definition of urban population.*[3] Since data were available for the city of Chicago as a unit, these were subtracted from the corresponding urban data resulting in a division of the population of Illinois into three groups: a, the city of Chicago, b, incorporated municipalities of 2,500 or more, exclusive of the city of Chicago, and c, all others, generally classifiable as rural. Based on these subdivisions it was possible to achieve correspondence of death data and population data, and therefore to calculate age-race-sex specific death rates for the three geographic areas.

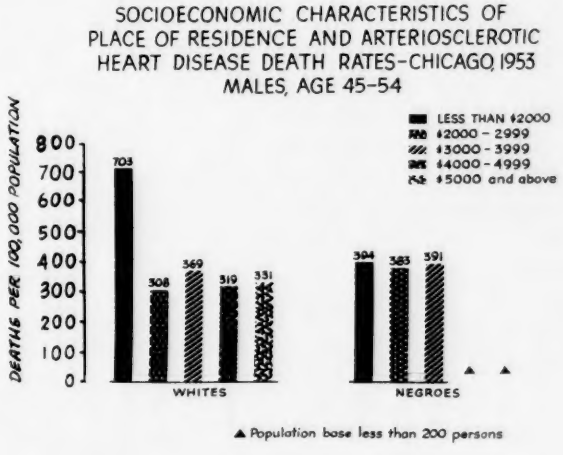

Fig. 1.

RESULTS

Chicago Death Rates by Age-Race-Sex-Census Tract of Residence (1953).— The death rates for arteriosclerotic heart disease (ASHD), for ASHD plus other myocardial degeneration, and for hypertensive disease for the age groups 45 to 54 and 55 to 64 are presented in Table I. The population sizes, also given in Table I, varied considerably, being very small for several groups. Specifically, for the nonwhite males and females of census tract groups IV and V the largest was only 208 persons. Among the whites, group I (census tracts with median income under $2,000) had the fewest persons in each age-sex group.

*There are several special types of communities which are also included in the 1940 definition of urban other than incorporated municipalities having a population of 2500 or more, but these were not represented in Illinois.

Data on Arteriosclerotic Heart Disease (ASHD—ISC 420)* revealed a relative uniformity of rates within a given age-race-sex group, with two consistent exceptions. For white males and white females in both 10-year age groups, census tract group I exhibited grossly higher death rates than any of the other groups included in the survey. (Table I and Fig. 1). The rates for ASHD and other myocardial degeneration combined (ISC 420+422) showed a similar pattern (Table I).

With respect to hypertensive disease, very few deaths were recorded in several of the groups. Under these circumstances, no consistent pattern of hypertensive death rates was discerned for the five tract divisions.

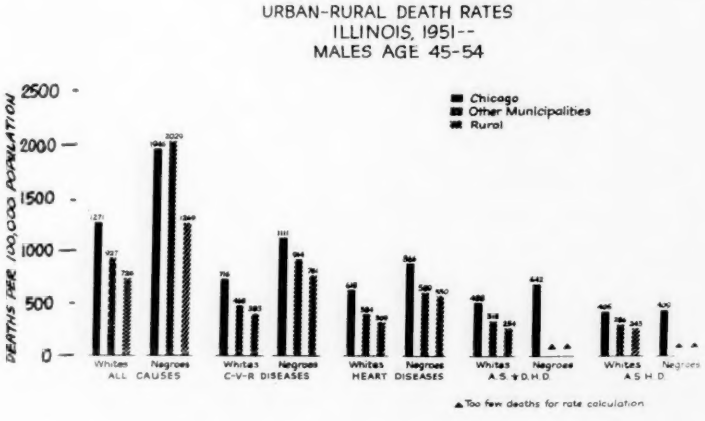

Fig. 2.—C-V-R diseases are the totals for the broad grouping of the cardiovascular-renal (CVR) diseases; AS&DHD is arteriosclerotic and degenerative heart disease (ISC categories 420+422); ASHD is arteriosclerotic heart disease (category 420).

Illinois Death Rates by Age-Race-Sex-Place of Residence (1951).—The population base for each age-race-sex-place of residence group was sizable, except for the municipal and rural nonwhite (Table II). The age-race-sex pattern of death rates† for each of the three geographic areas was, in general, qualitatively similar to that previously reported for Chicago.[1]‡ Quantitatively, death rates were generally lower in the municipal and rural areas than in Chicago. The rural rates tended to be lowest. This was true for both the broad categories and the specific cardiovascular-renal causes (Tables III-V and Fig. 2). The few exceptions were found in the 25 to 34 year age group, in the nonwhite female population, and in the cerebrovascular disease category (ISC 330-334) for the oldest age group.

*International Statistical Classification of Diseases, Injuries and Causes of Death, Sixth Revision, 1948, Code Number 420.

†The size of the population base and the number of deaths rendered impractical the calculation of non-white rates for specific causes, particularly for the rural population.

‡The rates for Chicago, itself, are, of course, identical in the two reports.

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TABLE I. AGE-RACE-SEX SPECIFIC DEATH RATES* FOR ASHD (I.S.C. 420), ASHD AND OTHER MYOCARDIAL DEGENERATION (I.S.C. 420 + 422), AND HYPERTENSIVE DISEASE (I.S.C. 440-447) FOR FIVE GROUPS OF CENSUS TRACTS CLASSIFIED ACCORDING TO MEDIAN INCOME† WITH THE CORRESPONDING POPULATION DATA. CHICAGO DEATHS 1953.

CAUSE (#I.S.C.)	CENSUS TRACT GROUP	AGE-RACE-SEX							
		45–54				55–64			
		W–M	NW–M	W–F	NW–F	W–M	NW–M	W–F	NW–F
420	I	703	394	267	220	1,446	1,004	433	697
	II	308	383	128	151	833	857	347	574
	III	369	191	109	———	915	1,292	375	998
	IV	319	———‡	72	———	876	———	354	———
	V	331	———	84	———	918	———	375	———
420 + 422	I	850	422	267	465	1,607	1,325	938	952
	II	395	535	156	321	1,006	1,095	439	831
	III	443	547	154	268	1,046	1,723	478	1,414
	IV	350	———	95	———	987	———	430	———
	V	384	———	101	———	995	———	414	———
440 – 447	I	———	191	———	196	80	256	———	302
	II	34	203	25	191	102	310	128	445
	III	41	195	41	536	103	517	131	998
	IV	38	———	28	———	113	———	85	———
	V	———	———	———	———	78	———	65	———
Population 1950	I	6,117	8,375	1,871	8,174	6,223	4,681	1,386	4,305
	II	26,608	21,143	24,292	19,937	22,560	10,963	19,575	10,110
	III	92,869	2,559	96,734	2,238	82,616	1,161	82,596	1,202
	IV	73,107	179	78,286	208	59,156	83	60,253	121
	V	11,187	39	11,842	49	7,737	14	7,738	26

*Deaths per 100,000 population.
†Census tract grouping according to median income:
I less than $2000
II $2000—2999
III $3000—3999
IV $4000—4999
V $5000 and over
‡Less than 5 deaths were recorded for these categories

DISCUSSION

The data for Chicago subdivided according to census tracts strongly indicate that coronary disease and hypertensive disease death rates are uniform within a given age-race-sex stratum of the Chicago population, irrespective of income. There is certainly no tendency for rates to be lower in the lower income groups.* These findings are consistent with those of the previous paper and other recent reports, indicating similar patterns for different occupation groups within a given age-race-sex stratum.[1]

These observations suggest that both coronary and hypertensive disease frequently cause death among all strata of the American middle-aged urban population, irrespective of income, occupation and socioeconomic stratification—

*If anything, the data indicate that for the whites the rates may be higher in the population with median family income less than $2000 per year. This particular finding should be regarded as tentative, since the census tracts of group I include the highly transient population of Chicago's 'Skid Row' (16 tracts out of a total of 46).

a conclusion in accord with limited data from other sources.[1] Apparently deaths from these diseases occur at least as frequently, and possibly even more frequently, in the lower income-manual laborer strata of the middle-aged population. The possible significance of these findings for the dietary-metabolic theory of atherogenesis was briefly discussed in the previous paper.[1]

TABLE II. POPULATION DATA BY AGE-RACE-SEX FOR THE STATE OF ILLINOIS DIVIDED INTO THE CITY OF CHICAGO, OTHER MUNICIPALITIES OF 2,500 OR MORE, AND ALL OTHER (RURAL), 1950.

SEX-RACE	POPULATION	AGE			
		25–34	35–44	45–54	55–64
W–M	Chicago	255,515	234,525	207,265	175,310
	Municipalities	208,321	200,160	172,917	137,799
	Rural	159,252	151,428	126,589	108,267
NW–M	Chicago	47,055	40,530	33,035	17,225
	Municipalities	9,583	8,764	6,456	4,145
	Rural	3,463	2,772	2,364	1,732
W–F	Chicago	272,525	255,150	211,600	173,020
	Municipalities	220,406	210,255	180,683	142,766
	Rural	159,662	141,450	117,918	98,690
NW–F	Chicago	54,885	44,745	30,570	15,685
	Municipalities	11,281	9,023	7,042	4,252
	Rural	2,086	2,173	1,910	1,366

TABLE III. AGE-SPECIFIC DEATH RATES* FOR SEVERAL SUMMARY GROUPS OF CAUSES BY PLACE OF RESIDENCE IN ILLINOIS (WHITE POPULATION), 1951.

CAUSE	POPULATION	MALE				FEMALE			
		AGE							
		25–34	35–44	45–54	55–64	25–34	35–44	45–54	55–64
Diseases of the Heart	Chicago	34	181	618	1,554	13	59	213	657
	Municipalities	13	106	384	1,044	9	36	135	492
	Rural	14	75	309	800	11	28	116	373
CV	Chicago	41	205	693	1,779	15	80	288	854
	Municipalities	17	123	449	1,282	16	51	205	684
	Rural	18	93	371	979	16	38	182	556
CVR	Chicago	47	213	716	1,822	19	85	297	891
	Municipalities	23	131	468	1,317	20	59	218	718
	Rural	24	101	383	1,018	18	42	192	585
All Causes	Chicago	168	483	1,271	2,989	99	276	690	1,634
	Municipalities	183	359	927	2,290	111	226	534	1,388
	Rural	167	305	726	1,714	95	190	466	1,129

*Deaths per 100,000 population.

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The Chicago-other municipalities-rural data indicate that death rates for the cardiovascular-renal diseases, including coronary disease and hypertensive disease, are lower outside the metropolis. This appears to be particularly true in the rural areas. These findings are also consistent with limited data available from other sources.[5-7] It does not appear that these patterns, particularly the higher rates for Chicago as against those of the municipal and rural areas, are factitious byproducts of concealed bias. Thus, the fact that these differences hold for all causes and the broad grouping of the cardiovascular-renal diseases, as well as for the specific causes (A.S. & D.H.D. and A.S.H.D.)—and the fact that the usual sex differential obtains—both mitigate strongly against the possibility that differences in diagnosis between urban and rural areas are responsible for the different patterns. The one possibility, that significant numbers of rural and municipal residents die in Chicago with inaccurate registration

TABLE IV. AGE-SPECIFIC DEATH RATES* FOR SEVERAL SUMMARY GROUPS OF CAUSES BY PLACE OF RESIDENCE, IN ILLINOIS (NONWHITE POPULATION), 1951.

CAUSE	POPULATION	MALES				FEMALES			
		AGE							
		25–34	35–44	45–54	55–64	25–34	35–44	45–54	55–64
Diseases of the Heart	Chicago	83	289	866	1,945	51	239	697	1,435
	Municipalities	42†	183	589	1,616	62	155	724	1,199
	Rural	29†	72†	550	231†	96†	276	105†	293†
CV	Chicago	100	363	1,078	2,450	78	329	916	1,996
	Municipalities	42†	274	883	2,388	80	266	1,037	1,576
	Rural	87†	108†	761	346	96†	414	262	805
CVR	Chicago	111	382	1,111	2,525	91	342	955	2,098
	Municipalities	73	285	914	2,485	98	344	1,136	1,787
	Rural	116†	108†	761	346	96†	414	262	1,098
All Causes	Chicago	487	883	1,946	4,261	337	735	1,652	3,054
	Municipalities	438	696	2,029	4,439	319	842	1,889	3,010
	Rural	318	469	1,269	1,270	240	552	681	1,537

*Deaths per 100,000 population.
†Rate based on less than 5 deaths.

of their addresses on the death certificates is unlikely. The absence of substantial differences, in general, for the lowest age group seems to indicate that this is improbable, unless some factor is operating differently for the different age groups as to resident registration. On the other hand, the differences between death rates for the rural areas and other municipalities are more open to question in view of the chance for improperly describing a deceased farm resident as a resident of his nearest municipality.* Nevertheless, this type of error would necessarily need to be of large magnitude to explain the differences observed and would not negate those between Chicago and the remainder of the state of Illinois.

*Here this would involve only those incorporated municipalities of 2,500 or more.

These findings on cardiovascular-renal mortality, and particularly on coronary heart disease mortality, pose problems with respect to the dietary-metabolic theory of atherogenesis. There is no evidence to show sizable differences in diet patterns among these areas,[1,4,7,8] nevertheless the rates appear to be considerably lower in the rural areas. Differences in habitual physical activity of work, in populations ingesting a potentially atherogenic diet, may play a significant

TABLE V. AGE-SPECIFIC DEATH RATES* FOR SEVERAL CVR CAUSES OF DEATH BY PLACE OF RESIDENCE IN ILLINOIS (WHITE POPULATION), 1951.

CAUSE (#I. S. C.)	POPULATION	MALE				FEMALE			
		AGE							
		25–34	35–44	45–54	55–64	25–34	35–44	45–54	55–64
330–334	Chicago	5	18	62	184	2	11	67	162
	Municipalities	3	13	52	210	4	11	60	165
	Rural	3	14	51	151	3	7	54	163
420	Chicago	14	107	405	1,051	3	17	94	340
	Municipalities	6	78	286	804	3	9	63	293
	Rural	8	54	243	612	3	9	53	211
422	Chicago	6	30	83	227	2	13	36	120
	Municipalities	1	7	32	91	1	7	13	53
	Rural	1	5	11	62	2	6	17	55
420 + 422	Chicago	20	137	488	1,278	4	30	130	460
	Municipalities	7	85	318	896	4	15	75	346
	Rural	9	59	254	674	5	15	70	266
430 – 434	Chicago	4	10	38	90	2	5	11	26
	Municipalities	1	3	10	29	1	1	7	16
	Rural	0	5	12	29	1	2	10	14
440 – 447	Chicago	2	15	57	152	1	8	42	145
	Municipalities	1	10	37	124	1	9	28	142
	Rural	1	4	21	75	1	8	26	73
590 – 594	Chicago	7	9	23	43	3	6	9	38
	Municipalities	5	9	20	36	4	7	13	34
	Rural	6	9	12	39	3	4	10	28
260	Chicago	2	4	9	39	0	5	19	76
	Municipalities	3	5	8	26	2	5	13	48
	Rural	2	5	5	31	1	3	15	44

*Deaths per 100,000 population.

role in accounting for death rate differentials.[9] Presumably these groups exhibit such differences with the rural areas perhaps tending toward higher levels of physical activity. If this be in any way responsible, it has an effect not exhibited by the people engaged in different occupations within Chicago.[1] It may be further suggested that a lower prevalence of heavy smoking among rural residents contributes to their lower coronary disease mortality rates.[7] The presumed lesser "stress and strain" of life in smaller towns and rural areas may also be invoked to account for their lower rates of coronary disease mortality. Reliable data are

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not available concerning either the relative degrees of "stress and strain" in urban versus rural populations or their relationship to coronary disease. In general, the extant data are so limited as to compel the conclusion that these "explanations" are all speculative and hypothetical. Further work on the epidemiology of cardiovascular-renal diseases in urban versus rural areas and the factors responsible for verified patterns of disease would appear to be a fruitful avenue for research.

In noting these urban-rural differences, it is important to observe further that even these rural rates for coronary heart disease are relatively high, both in relation to rates for all causes, and in relation to rates for this disease in other populations.[4] Thus, coronary disease is a major cause of death in middle-age in rural Illinois. Based on the known facts concerning dietary patterns in U.S. rural areas (1,4,8), this finding may be interpreted as consistent with, although of course not yielding definitive proof of, the nutritional-metabolic theory of atherogenesis.

SUMMARY

An epidemiologic analysis was accomplished of cardiovascular-renal (C-V-R) mortality rates by age-race-sex-place of residence for the city of Chicago (1953) and the state of Illinois (1951). The place of residence classification was based on income for the city of Chicago, and urban-rural differentiation for the state of Illinois. No significant differences were noted in C-V-R mortality rates—including rates for arteriosclerotic heart disease (ASHD)—in the Chicago population groups stratified by income. Residents of rural Illinois exhibited lower C-V-R and ASHD mortality rates than Chicagoans. However, these categories remained leading causes of death in middle-aged residents of rural Illinois.

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EPIDEMIOLOGIC STUDIES ON CARDIOVASCULAR-RENAL DISEASES: III. ANALYSIS OF MORTALITY BY AGE-SEX-NATIONALITY

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THE TWO previous papers of this series presented findings on cardiovascular-renal mortality in the Chicago and Illinois middle-aged populations, stratified by age-sex-race-occupation-income-place of residence (urban-rural).[1,2] The present study extends this analysis to encompass mortality data for the Chicago area foreign-born whites by nationality; for the Japanese-American, Chinese-American, and American-Indian residents of the entire United States; and for several foreign countries corresponding to some of the nationality groups of the Chicago-area foreign-born.

METHODS

For the calculation of death rates for the Chicago area foreign-born whites by country of nativity, the population base was obtained from the published reports of the 1950 census.[3] The age-sex-nationality specific population data were available for the Chicago Standard Metropolitan Area (SMA), but not for the City of Chicago itself. The Chicago SMA embraces six counties: Cook, Dupage, Kane, Lake, and Will Counties in Illinois and Lake County, Indiana. Mortality data for the year 1951† were available for the five Illinois counties. It was therefore necessary to obtain a population base for the Chicago SMA, less Lake County, Indiana. Age-sex-nationality data were not available for Lake County, Indiana per se. Therefore, the size of the desired population was estimated, based on the availability of totals (nonage and nonsex specific) for

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†The year 1951 was the year reported on in the preceding papers.

the foreign-born by nationalty for Lake County, Indiana. It was assumed that each foreign-born nationality group exhibited an age-sex distribution in Lake County, Indiana similar to that exhibited in the Chicago SMA as a whole. This permitted estimates of the age-sex-nationality specific population data for Lake County. The subtraction of these estimates from the corresponding totals for the Chicago SMA yielded the needed population base. The age groups considered were two ten-year spans, 45 to 54 and 55 to 64 years of age. The upper limit of 65 years is in accord with that of the two previous papers. The lower limit was selected because of the small magnitude of the foreign-born population below age 45.

The Bureau of Statistics, Illinois Department of Public Health, accomplished a special analysis of the deaths occurring in 1951 in order to make available age-sex-nationality specific mortality data for these foreign-born.

The National Office of Vital Statistics, United States Public Health Service made available age-sex-cause specific mortality data for 1949, 1950, 1951, and 1952 in the Japanese-American, Chinese-American and American-Indian populations. Population bases for the calculation of mean annual death rates for these 4 years were obtained from published reports of the 1950 census.[4]

Age-sex specific death rates for a number of foreign countries were calculated from the published data of the World Health Organization.[5] It was possible to obtain death rates for diseases of the heart and for all causes for Ireland, Norway-Sweden, and England–Wales for 1951. The rates for Italy and the German Federal Republic are for 1952; the rates for Austria are for 1953.

RESULTS

Chicago Mortality Rates by Age-Sex-Nationality of the Foreign-Born.—For all categories examined, age-sex specific data for the total foreign-born population yielded rates which were fairly similar to those for the total white population (including the foreign-born) of the Chicago SMA (Illinois portion) (Table I). In general, the rates for the foreign-born males were slightly to moderately lower than for all Chicagoans. The differences in male death rates were evident for broad disease groupings and for some specific categories, e.g. hypertensive disease and particularly arteriosclerotic heart disease. These differences were generally not evident for females.

Further analysis by individual nationalities revealed sizeable differences among the foreign-born in age-sex specific rates for all causes, for the broad cardiovascular-renal (CVR) group and for specific heart diseases, including coronary disease (ISC 420) (Tables II to V). Thus for males aged 45 to 54 and 55 to 64 the Italian and Austrian nationalities in particular exhibited consistently low rates relative to all foreign-born and all whites, whereas the Polish and Irish had relatively high rates, higher than those for all whites. In the 55 to 64 year old males, low rates were also noted for the Norwegian-Swedish group, and high rates for the Russian* (Table III).

*According to a survey of the Jewish population of the city of Chicago, about four-fifths of the Russian-born population is Jewish.[6]

For the women of these two age groups the pattern was slightly different (Tables IV and V). The English-Welsh, Norwegian-Swedish, and German groups tended to have low rates, whereas the Poles tended to have conspicuously high rates, compared to all foreign-born and all whites.

Mortality Rates by Age and Sex for Several Foreign Countries.—Age-sex specific death rates for diseases of the heart and for all causes are given in Table VI for several foreign countries and for the corresponding foreign-born population of the Chicago area. Also included for comparison are rates for the white population of the United States in 1950 and of the Chicago SMA (Illinois portion) in 1951. Using published material readily available it was possible to obtain information corresponding to six of the groups used in the previous comparisons. These were Austria, Italy, the German Federal Republic, Norway and Sweden, Ireland, and England and Wales.

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TABLE I. DEATH RATES* BY AGE, SEX, AND CAUSE FOR THE WHITE POPULATION AND THE TOTAL FOREIGN-BORN POPULATION OF THE ILLINOIS PORTION OF THE CHICAGO SMA, 1951.

CAUSE OF DEATH AND CODE NUMBER†	POPULATION‡	AGE AND SEX			
		45–54		55–64	
		M	F	M	F
Diabetes mellitus (260)	Foreign born	10	20	31	89
	All white	9	16	35	71
Vascular lesions affecting the central nervous system (330–334)	Foreign born	59	61	154	189
	All white	59	63	180	164
Chronic rheumatic heart disease (410–416)	Foreign born	26	21	27	25
	All white	29	31	34	35
Arteriosclerotic heart disease (ASHD) including coronary disease (420)	Foreign born	317	91	862	406
	All white	379	83	970	336
Other myocardial degeneration (422)	Foreign born	70	36	183	105
	All white	66	28	188	101
ASHD and other myocardial degeneration (420+422)	Foreign born	387	126	1,044	511
	All white	445	111	1,158	437
Other diseases of the heart (430–434)	Foreign born	21	9	69	26
	All white	32	11	73	23
Hypertensive heart disease (440–443)	Foreign born	42	29	113	143
	All white	45	32	122	124
Other hypertensive disease (444–447)	Foreign born	1	0	8	11
	All white	8	4	10	12
Hypertensive disease (440–447)	Foreign born	43	29	120	155
	All white	53	36	132	136
Nephritis and nephrosis (590–594)	Foreign born	19	8	45	36
	All white	20	10	41	36
Diseases of the heart (410–443)	Foreign born	482	185	1,261	707
	All white	555	186	1,395	625
Cardiovascular diseases (330–334, 400–468)	Foreign born	548	249	1,449	932
	All white	630	258	1,612	820
Cardiovascular-Renal diseases (330–334, 400–468, 590–594)	Foreign born	566	256	1,494	969
	All white	650	268	1,653	898
All causes (001–999)	Foreign born	1,065	622	2,593	1,685
	All white	1,151	636	2,727	1,585

*Deaths per 100,000 population.

†International Statistical Classification of Diseases, Injuries, and Causes of Death, Sixth Revision, 1948.

‡F B—The foreign-born population of the Illinois portion of the Chicago SMA.
All White—The white population of the Illinois portion of the Chicago SMA.

TABLE II. DEATH RATES* FOR THE FIVE ILLINOIS COUNTIES OF THE CHICAGO (SMA) AND FOR THE FOREIGN-BORN OF THESE COUNTIES BY COUNTRY-OF-ORIGIN. (MALES AGE 45 TO 54, 1951).

CAUSE	COUNTRY OF ORIGIN												
	ENGLAND AND WALES	IRELAND	NORWAY AND SWEDEN	GERMANY	ITALY	RUSSIA	POLAND	CZECH.	AUSTRIA	EAST CENTRAL EUROPE	ALL OTHER	FOREIGN-BORN CHICAGO SMA	ALL WHITE CHICAGO SMA
330 – 334	———	167	82	54	57	40	57	73	90	59	45	59	59
420	349	474	300	283	248	440	460	341	210	398	259	317	379
420 + 422	349	697	355	316	324	507	563	341	299	499	328	387	445
440 – 447	39	84	14	65	10	27	92	24	60	55	41	43	53
410 – 443	349	864	450	424	381	600	689	414	419	575	423	482	555
CV	388	1,031	545	490	448	654	758	487	509	643	468	548	630
CVR	388	1,059	573	501	467	667	793	511	509	664	484	566	650
All causes	698	1,812	1,091	1,001	847	1,094	1,470	1,047	868	1,192	981	1,065	1,151
Population size	2,580	3,588	7,334	9,190	10,502	7,496	8,705	4,106	3,342	23,649	24,361	81,204	305,893

*Deaths per 100,000 population.

TABLE III. DEATH RATES* FOR THE FIVE ILLINOIS COUNTIES OF THE CHICAGO (SMA) AND THE FOREIGN-BORN BY COUNTRY OF ORIGIN. (MALES AGE 55 TO 64, 1951).

CAUSE	COUNTRY OF ORIGIN												
	ENGLAND AND WALES	IRELAND	NORWAY AND SWEDEN	GERMANY	ITALY	RUSSIA	POLAND	CZECH.	AUSTRIA	EAST CENTRAL EUROPE	ALL OTHER	FOREIGN-BORN CHICAGO SMA	ALL WHITE CHICAGO SMA
330 – 334	38	154	162	164	148	200	181	223	182	191	111	154	180
420	866	1,110	663	835	678	1,127	979	811	568	945	851	862	970
420 + 422	1,054	1,356	799	1,013	794	1,286	1,200	906	772	1,131	1,059	1,044	1,158
440 – 447	113	123	108	110	156	84	158	80	91	123	107	120	132
410 – 443	1,318	1,665	988	1,190	1,012	1,454	1,471	1,081	954	1,356	1,270	1,261	1,395
CV	1,355	1,880	1,151	1,437	1,199	1,707	1,697	1,304	1,159	1,585	1,404	1,449	1,612
CVR	1,355	2,065	1,151	1,464	1,246	1,718	1,769	1,351	1,249	1,641	1,434	1,494	1,653
All causes	2,146	3,422	2,084	2,682	2,087	2,761	2,997	2,496	2,317	2,799	2,573	2,593	2,727
Population size	2,656	3,244	7,388	7,308	12,841	9,490	22,159	6,290	4,402	42,341	29,852	105,630	249,327

*Deaths per 100,000 population.

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TABLE IV. DEATH RATES* FOR THE FIVE ILLINOIS COUNTIES OF THE CHICAGO (SMA) AND THE FOREIGN-BORN BY COUNTRY OF ORIGIN. (FEMALES AGE 45 TO 54, 1951).

CAUSE	COUNTRY OF ORIGIN												
	ENGLAND AND WALES	IRELAND	NORWAY AND SWEDEN	GERMANY	ITALY	RUSSIA	POLAND	CZECH.	AUSTRIA	EAST CENTRAL EUROPE	ALL OTHER	FOREIGN-BORN CHICAGO SMA	ALL WHITE CHICAGO SMA
330 – 334	112	52	——	64	36	55	70	38	26	55	89	61	63
420	——	78	39	38	61	55	140	77	130	106	131	91	83
420 + 422	——	129	39	64	61	83	210	115	156	154	179	126	111
440 – 447	——	78	——	13	12	28	70	——	26	41	26	29	36
410 – 443	38	259	58	102	121	138	303	153	260	230	221	185	186
CV	150	311	58	165	158	208	373	191	286	288	315	249	258
CVR	150	311	58	165	158	208	404	191	286	302	326	256	268
All causes	375	621	423	521	400	470	823	574	755	682	756	622	636
Population size	2,670	3,864	5,197	7,863	8,255	7,228	12,878	5,225	3,841	29,172	19,037	76,058	307,022

*Deaths per 100,000 population.

TABLE V. DEATH RATES* FOR THE FIVE ILLINOIS COUNTIES OF THE CHICAGO (SMA) AND THE FOREIGN-BORN BY COUNTRY OF ORIGIN. (FEMALES AGE 55 TO 64, 1951).

CAUSE	COUNTRY OF ORIGIN												
	ENGLAND AND WALES	IRELAND	NORWAY AND SWEDEN	GERMANY	ITALY	RUSSIA	POLAND	CZECH.	AUSTRIA	EAST CENTRAL EUROPE	ALL OTHER	FOREIGN-BORN CHICAGO SMA	ALL WHITE CHICAGO SMA
330 – 334	232	291	135	126	241	168	202	153	122	179	202	154	164
420	186	582	285	309	422	415	523	336	268	444	390	406	336
420 + 422	325	688	315	365	519	479	672	458	365	566	501	511	437
440 – 447	47	185	150	84	169	117	206	153	122	171	149	155	136
410 – 443	418	873	541	450	712	609	936	611	536	775	718	707	625
CV	651	1,191	721	632	989	803	1,181	779	682	987	959	932	820
CVR	697	1,243	766	646	989	829	1,243	794	682	1,028	1,002	969	898
All causes	1,348	1,958	1,367	1,236	1,725	1,451	2,083	1,543	1,267	1,783	1,725	1,685	1,585
Population size	2,152	3,780	6,657	7,119	8,290	7,720	20,835	6,547	4,105	39,207	20,750	87,955	240,600

*Deaths per 100,000 population.

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TABLE VI. DEATH RATES* FOR FOREIGN-BORN CHICAGO AREA† RESIDENTS (1951) AND FOR RESIDENTS OF THE COUNTRY OF THEIR BIRTH:‡ DISEASES OF THE HEART AND ALL CAUSES BY SEX AND AGE

	AUSTRIANS		ITALIANS		GERMANS		NORWEGIANS AND SWEDISH		IRISH		ENGLISH AND WELSH		U. S. WHITES	ILLINOIS PORTION OF CHICAGO SMA—WHITES
	CHICAGO	EUROPE	CHICAGO	EUROPE	CHICAGO	EUROPE	CHICAGO	EUROPE	CHICAGO	EUROPE	CHICAGO	EUROPE		
Males 45 to 54														
Heart disease	419	167	381	148	424	136	450	136	864	244	349	218	330	555
All causes	868	870	847	847	1,001	772	1,091	580	1,812	960	698	867	1,006	1,151
Females 45 to 54														
Heart disease	260	66	121	102	102	68	58	64	259	175	38	98	67	186
All causes	755	525	400	509	509	507	423	447	621	748	375	532	547	636
Males 55 to 64														
Heart disease	954	509	1,012	412	1,190	418	988	437	1,695	719	1,318	717	830	1,395
All causes	2,317	2,152	2,072	1,917	2,668	1,898	2,084	1,436	3,452	2,269	2,146	2,424	2,353	2,727
Females 55 to 64														
Heart disease	536	236	712	309	450	226	541	258	873	503	418	336	267	625
All causes	1,267	1,215	1,725	1,249	1,236	1,218	1,367	1,080	1,984	1,789	1,348	1,312	1,292	1,585

*Deaths per 100,000 population.
†The five Illinois counties of the Chicago Standard Metropolitan Area.
‡See text for dates of data on foreign countries.

For the males the death rates from diseases of the heart were without exception considerably lower in the Europeans, compared with their equivalent Chicago foreign-born groups, with all white Chicagoans, and with all white Americans. For the females, the death rates from diseases of the heart also tended to be lower in the Europeans than in their Chicago foreign-born equivalents and all Chicago whites. These rates for European women tended to be generally similar to those for all white American women.

Mortality Rates by Age and Sex for Japanese-Americans, Chinese-Americans and American-Indians.—Sex and cause specific death rates for two age groups are given in Table VII and VIII for Chinese, Japanese, Indian, white, and Negro residents of the United States. The rates for the various cardiovascular-renal diseases for the Indian and the white populations were in general quite similar. However, coronary disease rates were lower in Indian than white males.[7,8] The rates for the various cardiovascular-renal diseases for the Japanese were conspicuously lower than for whites, with the notable exception of the rate for vascular lesions affecting the central nervous system* (ISC 330-334).[9] Chinese men had coronary disease rates slightly lower than those for the white population. Their rates for cerebrovascular, hypertensive and all cardiovascular-renal diseases were higher than those for the white population, this pattern being similar to that exhibited by Negroes. These cardiovascular-renal rates were, however, less than those for Negroes.

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DISCUSSION

In general, foreign-born males as a group seem to have lower mortality rates than native-born whites in middle-age for heart diseases, particularly coronary heart disease. The differences in rates seem definite, but are moderate in degree, so that the occurrence of coronary heart disease is by no means rare in the former groups in middle-age in the United States. Moreover, at least some of the individual nationality groups among the foreign-born experience rates as high as the native-born. Further, the foreign-born male groups in the United States exhibit heart disease death rates in middle-age sizably greater than those for their equivalents in the countries of origin. This seems true for the several European nationalities studied and for the Japanese.[9-15,17] Viewed over-all, it would seem valid to suggest that the foreign-born in the United States—in their patterns of heart disease and coronary disease mortality—may exhibit residual features reflecting the influence of their countries of origin, but their characteristics as Americans seem to be predominant. This tentative inference is supported by the limited data available for the foreign-born on such factors as habitual diets, prevalences of obesity, heavy smoking, hypertension, hypercholesterolemia—factors associated with increased incidences of coronary heart disease.[10,11,16,17] Thus, it has been reported that Italian-Americans have dietary habits and serum cholesterol levels more similar to those of native-born Americans than to those of Italians in their native country.[10-14,17] Similarly, Nisei in Los Angeles have patterns of diet and cholesterolemia like those of other Californians.[15] (Here it should be noted that Nisei are native-born, their parents having been the immigrants).

*For a detailed discussion of the statistics on cerebrovascular diseases, cf. refs. 10 and 11.

TABLE VII. AGE-SEX-SPECIFIC DEATH RATES* FOR THE CARDIOVASCULAR-RENAL DISEASES FOR THE CHINESE, JAPANESE, INDIAN, WHITE, AND NEGRO AMERICANS.

CAUSE	POPULATION	AGE—SEX				CAUSE	POPULATION	AGE—SEX			
		45 TO 54		55 TO 64				45 TO 54		55 TO 64	
		M	F	M	F			M	F	M	F
330 — 334	Chinese	96	118	349	269	440 — 447	Chinese	118	118	299	218
	Japanese	55	106	230	200		Japanese	35	47	88	79
	Indian	69	65	182	153		Indian	46	69	132	133
	White	55	55	186	157		White	42	36	122	104
	Negro	213	249	523	571		Negro	222	247	480	529
410 — 416	Chinese	27	25	33	34	590 — 594	Chinese	47	25	87	50
	Japanese	8	7	4	14		Japanese	27	15	22	28
	Indian	18	12	24	23		Indian	32	30	55	73
	White	24	23	34	30		White	18	12	33	25
	Negro	23	23	44	33		Negro	59	63	123	120
420	Chinese	225	67	682	252	410 — 443	Chinese	404	236	1,181	487
	Japanese	137	40	391	140		Japanese	199	106	563	237
	Indian	203	75	419	217		Indian	336	224	741	473
	White	330	67	830	267		White	433	142	1,105	460
	Negro	261	168	598	414		Negro	625	525	1,434	1,216
422	Chinese	33	17	143	17	CV	Chinese	525	354	1,583	805
	Japanese	8	7	62	9		Japanese	262	220	808	441
	Indian	23	28	90	73		Indian	411	304	952	669
	White	21	11	75	42		White	504	207	1,336	642
	Negro	72	51	189	164		Negro	890	820	2,069	1,885
420 + 422	Chinese	257	84	825	269	CVR	Chinese	572	379	1,670	856
	Japanese	145	48	453	149		Japanese	289	242	822	469
	Indian	226	102	509	290		Indian	443	334	1,008	743
	White	351	77	905	309		White	522	219	1,370	666
	Negro	334	220	786	578		Negro	949	883	2,192	2,005
430 — 434	Chinese	10	8	37	0	All causes	Chinese	1,533	724	3,442	1,477
	Japanese	8	7	11	0		Japanese	712	487	1,761	986
	Indian	37	41	71	43		Indian	1,489	1,162	2,484	2,155
	White	18	7	43	18		White	1,006	547	2,353	1,292
	Negro	59	47	120	91		Negro	1,913	1,575	3,775	3,105

*Deaths per 100,000 population per year.
Rates are 4-year averages (1949-50-51-52) for Chinese, Japanese and Indian.
White and Negro rates are for the single year 1950.

Recent living population studies by our group on the middle-aged employees of a Chicago utility company also failed to reveal gross differences between native-born and foreign-born in such parameters as prevalences of obesity, hypertension, and hypercholesterolemia.[16,17] Coronary heart disease incidence rates were significantly lower in the foreign-born. This was particularly evident for the foreign-born Irish, the main specific nationality group—in contrast to the foregoing mortality findings for the foreign-born Irish of Chicago as a whole. Thus, coronary disease incidence rates were 74, 30, and 34 per 1,000 per 4 years (1954-1957) for native-born, foreign-born Irish and all foreign-born males age 50 to 59 in this utility company population. The reasons for this apparent difference remain to be elucidated.

TABLE VIII.—POPULATION SIZE

	45 TO 54		55 TO 64	
	M	F	M	F
Chinese	12,248	2,968	7,517	1,490
Japanese	6,395	6,826	6,843	5,378
Indian	14,159	12,732	9,479	7,507
White	7,669,900	7,867,335	6,051,895	6,176,890
Negro	768,945	796,195	459,240	440,965

In conclusion, it would seem valid to suggest that the foreign-born in the United States are predominantly—but not wholly—Americanized in their mode of life, and this reflects itself in their mode of death. However, research on this problem has only begun, and the available data sorely need supplementation. Further pursuit of the apparently suggestive leads in this area—especially through living population studies—would seem to be very much in order, particularly since they may be quite enlightening with respect to intriguing problems of the etiology of coronary heart disease.

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SUMMARY

Death rates due to cardiovascular-renal and heart diseases, including coronary heart disease, were moderately lower in middle-aged foreign-born than in all Chicago white males. These rates were considerably higher for the foreign born Chicago males than for age-matched men in the corresponding countries abroad. Coronary heart disease death rates were higher in middle-aged white American males than in Japanese-Americans, and American-Indians, and Chinese-Americans.

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Epidemiology

MORBIDITY AND MARITAL STATUS

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DIVORCED persons have higher rates of illness than married persons. Illness rates of the married and the widowed vary only slightly when adjustment is made for age. Women who have never married show lower rates of illness than married women. These are some of the findings from a study of morbidity by marital status based on data from the California Health Survey, a household sample survey of illness in the general population.

This study of morbidity by marital status was stimulated by an interest in determining whether morbidity data follow the same pattern as mortality data. A number of studies of mortality by marital status have shown that married persons have lower death rates than nonmarried persons. In one such study the rates appear to be lower in every adult age group and for almost every cause of death (See Table I).[1,2]

Certain differences should be noted between mortality and morbidity studies of marital status groups. Mortality studies deal with a definite event while morbidity is not so easily defined. However, the basis of rates in morbidity studies from a household sample survey may be more accurate than that of mortality studies. In computation of mortality rates, census reports are the source of the statement of marital status in the population, and death certificates are the source of the statement of marital status at death. Thus, the calculation of mortality rates (number of deaths/population) may be unreliable as the numerator and denominator come from different sources. In morbidity data from a household survey, however, the source of data is the same for the numerator and denominator of each rate.

MATERIALS AND METHODS

The data presented were collected for the California Health Survey in 1954 and 55. Each week for 52 weeks interviewers obtained information covering illness, population characteristics, and medical care from a sample population

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TABLE I. AVERAGE DEATH RATES BY MARITAL STATUS, AGE, AND SEX IN THE UNITED STATES, 1949 TO 1951

AGE IN YEARS	MALE					FEMALE				
	TOTAL*	SINGLE	MARRIED	WIDOWED	DIVORCED	TOTAL*	SINGLE	MARRIED	WIDOWED	DIVORCED
Under 20	3.4	3.4	1.6	2.0	2.3	2.5	2.6	1.0	4.8	1.6
20–24	1.9	2.2	1.5	5.7	3.4	1.0	1.2	0.9	3.4	1.7
25–34	2.2	3.6	1.7	8.6	5.8	1.4	2.2	1.2	4.1	2.6
35–44	4.3	8.5	3.6	12.1	11.8	2.9	3.9	2.6	6.2	4.5
45–54	10.7	17.8	9.3	21.6	23.2	6.5	7.0	5.7	10.3	8.1
55–59	20.0	30.0	17.8	30.4	36.5	11.4	11.5	10.2	14.8	13.8
60–64	29.0	41.0	25.8	39.5	48.6	17.5	16.6	15.7	20.7	21.1
65–69	41.1	55.0	36.5	50.0	66.1	26.0	24.8	23.5	28.1	33.1
70–74	60.4	78.8	54.3	69.1	91.9	43.2	42.3	39.0	44.8	58.2
75 and over	119.4	137.3	100.3	139.0	173.3	101.6	103.6	76.0	106.2	129.2

*Includes deaths for which marital status was not stated.

Note: Rates are the number of average annual deaths per 1,000 population in each specified group, enumerated as of April 1, 1950. Exclusive of deaths among armed forces overseas.

Source: National Office of Vital Statistics, Vital Statistics—Special Reports, Vol. 39, No. 7, p. 376.

of about 200 households throughout the State. The sample, which was representative of California's civilian, noninstitutionalized population, included about 30,000 persons during the year.

The measures of morbidity used in this study are those as defined in the Survey report.[3] Morbidity will be examined in terms of incidence (the frequency of acute and chronic illness which began during the 4-week period preceding interview) and prevalence (the frequency of existing chronic conditions during the year). We shall also examine days of disability and hospitalizations as measures of severity of conditions.

These measures will be used to analyze health patterns of each marital status group by sex, for persons aged 25 through 74 years, avoiding the extremes of youth and old age. In addition to crude rates, age-adjusted rates will be presented, since age is a very important factor in morbidity rates. Analysis by specific age groups will not be presented since the number of persons in many of the categories was too small to exhibit stable rates (See Appendix Table A).

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CALIFORNIA MARITAL STATUS PATTERNS*

It was not until about 1940 that the marital status composition of California's population came to resemble that of the United States as a whole. The picture for females had never been very different from that of the nation, but the proportion of males in California who remained unmarried had been higher. Beginning in the early 1940's the proportion of married persons in California's population under 35 years rose substantially, as it did in the nation in general. This increase was especially great for males at ages 20 through 39 and almost as great for females 15 through 29. By 1954, 71 per cent of all Californians 15 years and over were married, as compared to 61 per cent in 1940.

The proportion of widowed persons (male and female) in California has for many years been about the same as that in the United States as a whole. The proportion widowed in California has changed only slightly from 1900 to 1950; the per cent widowed was 8.1 in 1900 and was 8.2 in 1950. As in the United States as a whole, the proportion of widowed females has always been larger than the proportion of widowed males. This is to be expected since women live longer than men and also tend to marry men older than themselves.

The proportion of divorced persons in California's population, however, has been approximately twice as high as in the nation as a whole in each census year since 1900. In 1950 the per cent divorced in California was 4.3 as compared to 2.2 in the United States. The proportion of divorced persons in California's population has increased more than fivefold since 1900. In that year, the proportion of divorced Californians was 0.8 per cent, contrasted with 4.3 per cent in 1950. Some of this increase may be attributed to more complete reporting. In addition, as evident in Fig. 1, the per cent of population "at risk" (i.e. the married) has greatly increased during this period. As with the widowed, there is a larger proportion of divorced females than divorced males.

*Data in this section are from Thompson.[5]

Fig. 1 shows the per cent distribution of Californians 15 years and over by marital status and sex from 1900 to 1954.

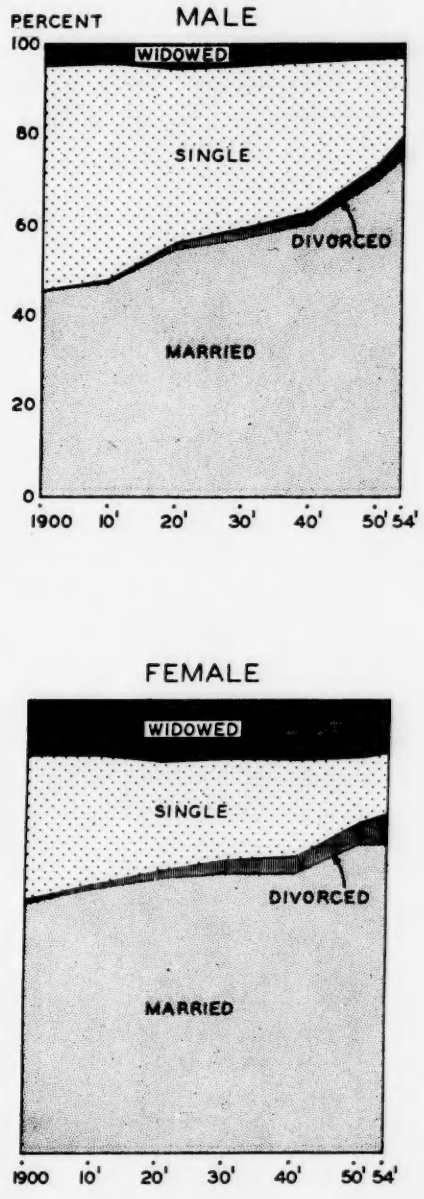

Fig. 1.—Per cent of population 15 years and over by marital status and sex, California, 1900-1954.

CALIFORNIA HEALTH SURVEY POPULATION

Selected Characteristics.—

Race: The marital status* composition of the various racial groups differs considerably. In Appendix Table B "Spanish Surname" has been separated from the rest of the "white" group because of possible difference in morbidity patterns.

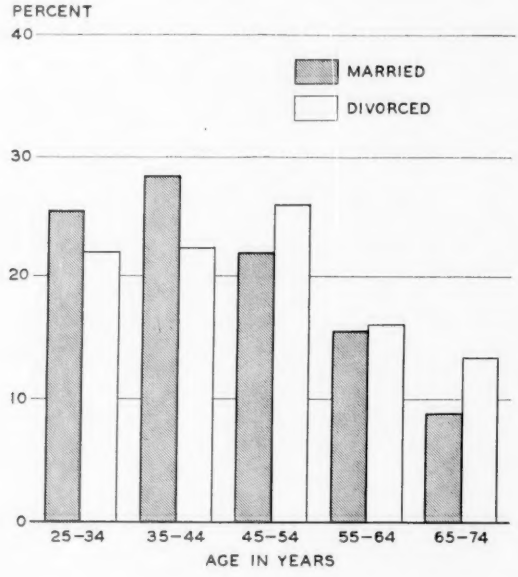

Fig. 2.—Per cent distribution of married and divorced males, by age, 25 to 74 years.

The Negro group exhibits a lower percentage married and a higher percentage divorced than does the white group. The per cent widowed is similar throughout the racial groups, while the "other" group appears to exhibit a higher proportion of "never married" males than do the white, Spanish Surname, or Negro groups.

Family income: Because income seems to be related to morbidity, an examination of the economic status of the Survey group may be helpful. Divorced persons tend to have lower incomes than the married. The proportion of persons in the low family income bracket (under $2,000) in the divorced group is over three times the proportion of low family income persons in the married group. The proportion of persons with low income in the never married group is also over three times that of the married group. The effect of age upon individual income may be important in these differences, as individual income increases with age up to a certain point and then decreases. Our data do not include

*In this study persons who had separated from their spouses or whose marriages had been annulled were included with divorced because of their probable similarity of living patterns.

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information about individual income. Since married people generally tend to live in households of 3 or more persons, the family income is divided among a greater number of persons than is the case with the nonmarrieds, who tend to live in one- or two-person households. The proportion of persons with low family income is almost five times as great in the widowed group as in the married group. This difference might be explained in part by the older age of the widowed population (See Appendix Table C).

Health insurance: The married group has a higher proportion of persons covered by health insurance than does the divorced group. The widowed group has the least health insurance coverage, perhaps because of the difficulty older persons have in obtaining health insurance. The only marital status group showing a different pattern of health insurance for males and females is the never married group, in which 58 per cent of the females are covered, while only 42 per cent of the males report coverage (See Appendix Table D).

MORBIDITY BY MARITAL STATUS

In this section the morbidity patterns of the married group will be compared first to the divorced, then to the widowed, and then to the never married groups.

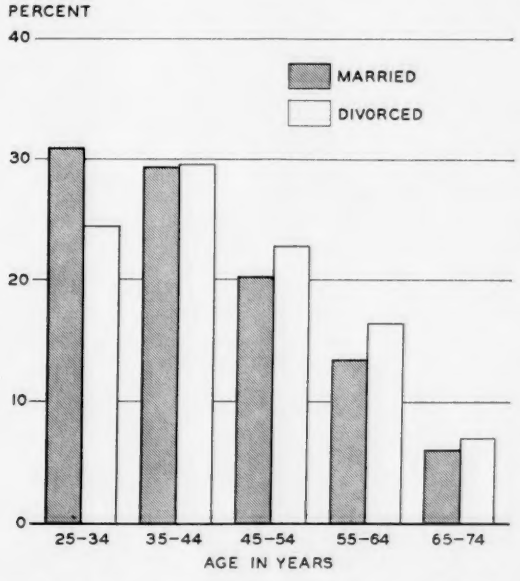

Fig. 3.—Per cent distribution of married and divorced females, by age, 25 to 74 years.

The married and divorced groups will be examined by crude rates and by application of adjusted rates.

I. Comparison of Married and Divorced Groups.—

Crude rates: In the California Health Survey data, the prevalence and incidence of chronic conditions and the days of disability from chronic illness

TABLE II. HEALTH AND MEDICAL CARE RATES FOR MARRIED AND DIVORCED GROUPS BY SEX AND AGE (25-74) IN THE CALIFORNIA HEALTH SURVEY*

	TOTAL		MALE		FEMALE	
	MARRIED	DIVORCED	MARRIED	DIVORCED	MARRIED	DIVORCED
Median age	42.56	44.88	43.69	47.12	41.44	43.67
Percent of population	79.4	6.6	83.7	5.2	75.3	7.9
Per year						
Incidence of illness per 1,000 persons						
Total	5,915	7,163	4,797	5,213	7,072	8,359
acute	2,704	2,821	2,483	2,535	2,925	2,990
chronic	3,211	4,342	2,314	2,678	4,147	5,369
disabling†	1,482	1,781	1,118	1,209	1,872	2,132
Days of disability per person						
Total	24.57	40.95	21.32	45.76	27.95	37.96
acute	5.59	6.11	3.90	4.81	7.28	6.89
chronic	18.98	34.84	17.42	40.95	20.67	31.07
Bed days of disability per persons						
Total	10.14	14.43	7.80	8.84	12.61	17.94
acute	2.21	2.47	1.56	1.56	2.99	3.12
chronic	7.93	11.96	6.24	7.28	9.62	14.82
Persons with chronic conditions (%)						
Total	60.0	66.2	55.3	62.9	65.0	68.2
causing some activity limitation	14.8	19.9	14.1	20.4	15.3	19.7
causing no activity limitation	45.2	46.3	41.2	42.5	49.7	48.5

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Chronic conditions per 1,000 persons						
Total	1,190	1,582	979	1,332	1,408	1,736
medically attended	946	1,197	780	976	1,116	1,333
disabling‡	257	380	209	323	307	415
Hospitalizations						
admissions per 1,000 persons						
including deliveries	114	102	82	85	146	113
excluding deliveries	86	89	82	85	90	92
days per 1,000 persons						
including deliveries	1,136	1,564	1,207	2,011	1,064	1,290
excluding deliveries	1,030	1,486	1,207	2,011	847	1,164
average length of stay days per admission						
including deliveries	9.99	15.29	14.72	23.77	7.26	11.40
excluding deliveries	12.02	16.68	14.72	23.77	9.46	12.67
Health insurance coverage (%)						
some	58.9	44.8	60.5	44.5	57.3	45.0
none	41.1	55.2	39.5	55.5	42.7	55.0

*Figures are subject to sampling variation.
†Disabling 1 or more days in past 4 weeks.
‡Disabling 1 or more days in past 12 months.
Note: For the interview period May 1954-April 1955 covering hospitalizations for May 1953-April 1955.
For definition of terms, see Health in California, Appendix C.
Source: State of California, Department of Public Health, Bureau of Chronic Diseases, California Health Survey.

increased with age, while incidence and days of disability from acute illness decreased slightly with age. Because of this pattern of age with respect to morbidity, it is necessary to consider the age distribution of the various marital status groups before comparing crude rates.

The divorced group exhibited a higher median age than did the married group, (women 2¼ years higher, men 3½ years higher). However, the age distribution of the two groups was similar in general form, as seen in Figs. 2 and 3.

In this paper the measures of morbidity, which encompass incidence, prevalence, disability, and hospitalization, will be referred to as health and medical care rates.

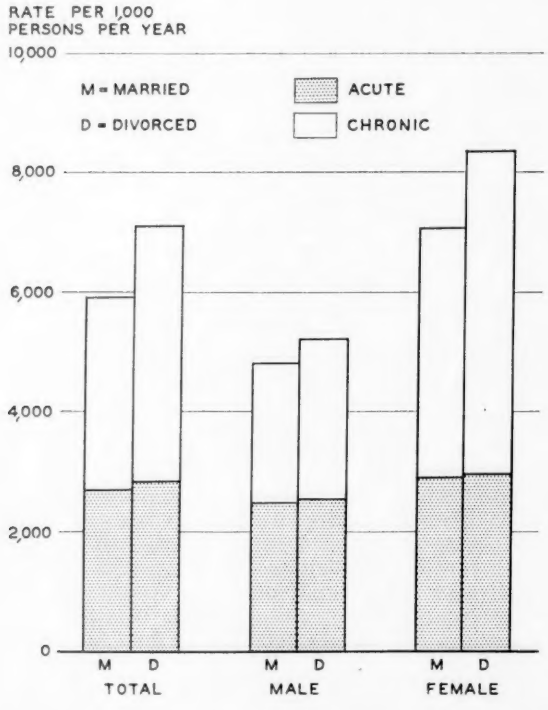

Fig. 4.—Incidence of illness for married and divorced, by sex, age 25 to 74 years.

Table II presents a broad view of health and medical care rates for the married and divorced groups. The married group shows consistently lower rates than the divorced group. In only one case—that of the rate of days of disability from acute illness in females—does the rate of the married exceed that of the divorced. For the divorced, the combined male and female rates of total days of disability, total incidence of illness, and total and disabling chronic conditions were found to be higher than corresponding rates for the married at the 5 per

cent level of significance. The differences between the two groups for hospital admissions and days of stay, as well as incidence of disabling illness, were within the limits of sampling variation.*

Although the incidence of total illness during the year was higher in the divorced group than in the married group, it is noteworthy that the rates of

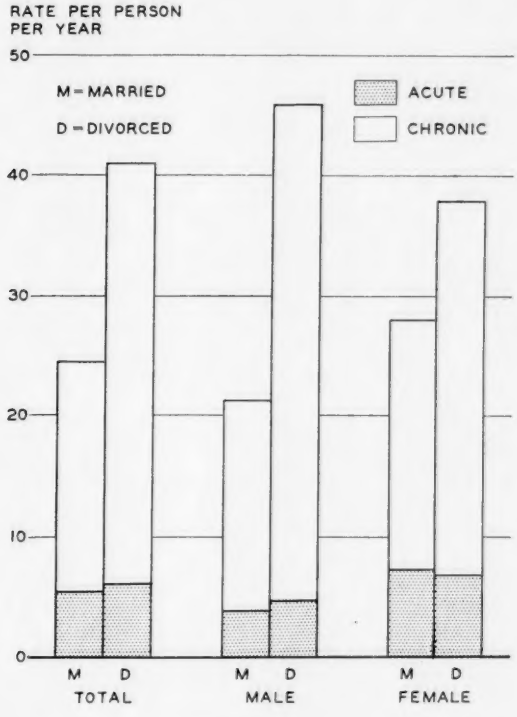

Fig. 5.—Days of disability per person per year for married and divorced, by sex, age 25 to 74 years.

acute illness were not very different in the 2 groups. The major difference occurred in the incidence of chronic illness, which was considerably higher for divorced persons than for married persons.

The days of disability per person per year were about two-thirds higher for divorced than for married of both sexes, and more than twice as high for divorced men as for married men. Since there was no great difference in days of disability from acute illness between married and divorced persons, disability from chronic illness accounted for almost the entire difference (See Fig. 5). Chronic illness accounted for 85 per cent of the days of disability for divorced persons, while only 77 per cent of the days of disability of the married group were attributed

*For discussion of sampling variation, see California State Department of Public Health publication Health in California, pages 65-68.

TABLE III. AGE-ADJUSTED RATES FOR SELECTED MEASURES OF MORBIDITY, IN BOTH MARRIED AND DIVORCED, BY SEX AND AGE (25-74) IN THE CALIFORNIA HEALTH SURVEY*

	AGE-ADJUSTED RATE					
	TOTAL		MALE		FEMALE	
	MARRIED	DIVORCED	MARRIED	DIVORCED	MARRIED	DIVORCED
Per year						
Incidence of illness per 1,000 persons Total	5,859	7,139	4,789	5,326	6,948	8,244
Chronic conditions per 1,000 persons Total	1,209	1,568	987	1,273	1,439	1,743
Days of disability per person Total	25.82	40.97	21.85	43.20	29.91	38.13

*Figures are subject to sampling variation.
Source: State of California, Department of Public Health, Bureau of Chronic Diseases, California Health Survey, 1954-1955.

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to chronic illness. Days of disability as used here is an all-inclusive term and offers a broad measure of morbidity. It includes not only acute illness, but also those chronic conditions which had continued over a long period of time and which had caused some disability during the survey period, as well as the disabling conditions which began during the 4-week period during which the survey was conducted.

A larger per cent of the divorced population than of the married reported one or more chronic conditions. In order to determine the effect of such a chronic condition upon a person, he was asked to indicate its impact upon his activities. The statements of degree of activity limitation provided another way of determining the severity of conditions. A larger per cent of the divorced persons reported conditions which caused some activity limitation.

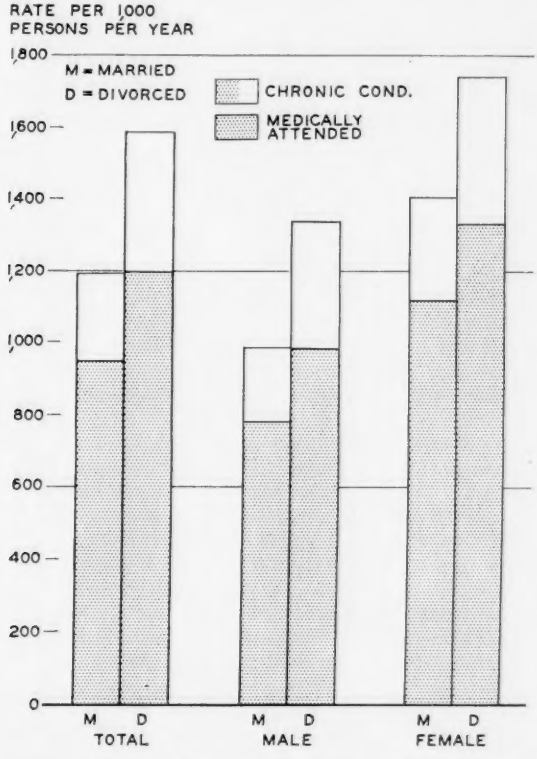

Fig. 6.—Chronic conditions per 1,000 persons per year for married and divorced, by sex, age 25 to 74 years.

The prevalence of chronic conditions followed a pattern similar to the other rates; divorced persons again showed higher rates than married persons (See Fig. 6).

The proportion of medically attended conditions to the total number of chronic conditions was slightly higher for the married than for the divorced (79.5 per cent of the married, 75.7 per cent of the divorced).

TABLE IV. AGE-ADJUSTED RATES FOR SELECTED MEASURES OF MORBIDITY BY RACE, INCOME, SEX, AND AGE (25-74) IN THE CALIFORNIA HEALTH SURVEY*

A. MORBIDITY BY RACE

	AGE-ADJUSTED RATE							
	MALE				FEMALE			
	WHITE	SPANISH SURNAME	NEGRO	OTHER	WHITE	SPANISH SURNAME	NEGRO	OTHER
Per year								
Incidence of illness per 1,000 persons Total	4,931	3,252	5,223	3,560	7,207	4,411	6,515	5,706
Chronic conditions per 1,000 persons Total	1,047	686	994	886	1,498	987	1,348	843
Days of disability per person Total	23.66	20.35	34.44	16.66	28.85	29.84	31.58	13.21

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B. MORBIDITY BY INCOME

	AGE-ADJUSTED RATE							
	MALE				FEMALE			
	LESS THAN $2,000	$2,000-4,999	$5,000 AND OVER	UNKNOWN	LESS THAN $2,000	$2,000-4,999	$5,000 AND OVER	UNKNOWN
Per year								
Incidence of illness per 1,000 persons								
Total	5,270	4,514	4,959	3,254	7,851	6,780	6,859	4,902
Chronic conditions per 1,000 persons								
Total	1,399	989	970	740	1,620	1,429	1,415	1,008
Days of disability per person								
Total	61.69	20.71	17.68	16.88	36.03	29.30	28.63	18.67

*Figures are subject to sampling variation.
Source: State of California, Department of Public Health, Bureau of Chronic Diseases, California Health Survey, 1954-1955.

TABLE V. AGE-RACE ADJUSTED RATES AND AGE-INCOME ADJUSTED RATES FOR SELECTED MEASURES OF MORBIDITY, MARRIED AND DIVORCED BY SEX AND AGE (25-74) IN THE CALIFORNIA HEALTH SURVEY*

A. MORBIDITY BY AGE AND RACE

	AGE-RACE ADJUSTED RATE			
	MALE		FEMALE	
	MARRIED	DIVORCED	MARRIED	DIVORCED
Per year				
Incidence of illness per 1,000 persons				
Total	4,783	5,270	6,966	8,384
Chronic conditions per 1,000 persons				
Total	987	1,292	1,440	1,772
Days of disability per person				
Total	21.87	41.60	29.71	38.23

B. MORBIDITY BY AGE AND INCOME

	AGE-INCOME ADJUSTED RATE			
	MALE		FEMALE	
	MARRIED	DIVORCED	MARRIED	DIVORCED
Per year				
Incidence of illness per 1,000 persons				
Total	4,802	5,234	7,044	8,125
Chronic conditions per 1,000 persons				
Total	1,001	1,212	1,456	1,708
Days of disability per person				
Total	23.03	34.09	30.13	36.10

*Figures are subject to sampling variation.

Source: State of California, Department of Public Health, Bureau of Chronic Diseases, California Health Survey, 1954-1955.

The pattern of medical care was also different in the 2 groups. Health insurance coverage was more common among married persons than among divorced persons. When deliveries were excluded, hospitalizations—both days in the hospital and rate of admissions—were higher for the divorced group. Married women had a higher rate of admissions when these were included. Examination of average length of hospital stay per admission for the married and divorced revealed that the divorced group stayed almost 40 per cent longer than the married group. The divorced males showed the greatest difference, with approximately 60 per cent longer average stay than married males.

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Adjusted rates:

Age Adjustment.—In order to minimize the effect of the differences in age upon the morbidity rates, age-adjusted rates have been computed for the married and divorced groups. The adjusted rates for the age group 25 through 74 years for each sex are to be used only to facilitate comparison and are not of value in terms of absolute magnitude.

Rates were adjusted to comparable age and sex groups of the entire California Health Survey population by the direct method of adjustment. Rates of incidence of illness, chronic conditions, and days of disability will be presented.

The consistent pattern of higher rates for divorced persons was again evident. Rates were higher for each measure as shown in Table III.

Age-Race Adjustment.—As noted earlier, the marital composition differed among the various racial groups. The rates were therefore examined by race for evidence of a pattern of differences with respect to this characteristic.

In Table IV, age-adjusted rates are presented by race for incidence of illness, chronic conditions, and days of disability. There does not appear to be a consistent pattern of higher rates for any one racial group. However, since there is variation, we applied an age-race adjustment to the rates for the married and divorced to determine if differences remained. The rates were adjusted by the indirect method of adjustment using specific rates of the California Health Survey population as a standard.

Table V presents age-race-adjusted rates for the married and divorced by sex. Note that these rates differ only slightly from the crude rates by marital status. The rates of the divorced remain higher for every measure. It may be concluded, therefore, that the racial factor is not influential in the difference in morbidity patterns between marital groups as reported in the Survey.

Age-Income Adjustment.—The indirect method of adjustment was used again to adjust for the family income composition of the marital groups. As shown in Table IV, the rates of the lowest family income group were higher for each measure.

Although the age-income-adjusted rates shown in Table V were again higher for the divorced group than for the married group, the rates did not vary by the same magnitude as did the crude rates or those adjusted for age alone. They were influenced in part by the family income composition of the group; however, this factor alone could not account for all of the difference between the rates of the divorced and those of the married group.

It is important to note that the relationship of morbidity to family income presents an obvious problem of identification of cause and effect. A circular pattern of low income and sickness often occurs; in some cases, low income is a result of sickness (and thus inability to work) and in other cases, sickness may be caused by poverty (inadequate food, shelter, medical care, etc.).[5] One may also note the effect of marital status upon income when low income is the result of sickness. A married person would often have a source of income in spite of illness when the marital partner works, while a divorced person would usually be his own sole support.

TABLE VI. HEALTH AND MEDICAL CARE RATES FOR WIDOWED AND AGE-ADJUSTED MARRIED AND NEVER MARRIED AND AGE-ADJUSTED MARRIED BY SEX AND AGE (25-74) IN THE CALIFORNIA HEALTH SURVEY*

	WIDOWED AND AGE-ADJUSTED MARRIED BY SEX				NEVER MARRIED AND AGE-ADJUSTED MARRIED BY SEX			
	MALE		FEMALE		MALE		FEMALE	
	MARRIED	WIDOWED	MARRIED	WIDOWED	MARRIED	NEVER MARRIED	MARRIED	NEVER MARRIED
Median age	43.69	64.06	41.44	62.45	43.69	38.95	41.44	40.28
Percent of population	83.7	2.6	75.3	11.0	83.7	8.4	75.3	5.7
Per year								
Incidence of illness per 1,000 persons								
Total	4,272	5,246	6,018	6,618	4,854	4,403	7,021	5,030
Disabling†	1,025	741	1,564	1,645	1,155	1,035	1,915	1,191
Days of disability per person								
Total	38.8	51.5	44.0	37.0	20.34	22.60	29.39	15.26
Bed days of disability per person								
Total	11.7	18.1	21.2	18.0	7.42	8.70	13.53	7.68
Persons with one or more chronic conditions (%)								
Total	64.5	71.1	69.9	72.1	54.6	53.4	65.2	58.3
Chronic conditions per 1,000 persons								
Total	1,290	1,645	1,658	1,711	954	1,005	1,423	1,202
Disabling‡	273	311	334	327	204	181	309	237
Hospitalizations (excluding deliveries)								
Admissions per 1,000 persons	103	140	88	78	77	79	86	80
Days per 1,000 persons	1,298	2,342	1,106	1,529	1,090	1,435	854	1,172
Average length of stay in days per admission	14.7	16.7	10.7	19.7	14.2	18.1	9.9	14.6

*Figures are subject to sampling variation.
†Disabling 1 or more days in past 4 weeks.
‡Disabling 1 or more days in past 12 months.
Note: For the interview period May 1954-April 1955 covering hospitalizations for May 1953-April 1955.
For definition of terms, see Health in California, Appendix C.
Source: State of California, Department of Public Health, Bureau of Chronic Diseases, California Health Survey.

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II. Comparison of Married and Widowed Groups.—

Comparison of the married and widowed groups was difficult because of the difference in age distributions of the two groups and the small size of the widowed group. The median age of widowed persons was about 20 years higher than that of married persons. The total crude rates were thus not comparable, because age is so important a factor in morbidity rates. In computing age-adjusted rates, the married group was found to have enough persons throughout the age range to provide fairly stable age-specific rates; however, the widowed group had sufficient concentration of persons only in the older age groups. Therefore, to facilitate comparison of the two groups, we have adjusted the morbidity rates of the married group to the population of the widowed (by sex) by the direct method of adjustment.

Table VI shows health and medical care rates for the widowed and the same rates, age-adjusted, for the married. No consistent pattern is obvious.

Incidence rates for total illness were higher in the widowed group than in the married group. Incidence of disabling illness, however, was higher for married males than for widowed males.

Days of disability per person per year from both acute and chronic illness were higher for married females than for widowed females.

The proportion of widowed men reporting one or more chronic conditions was higher than that of married men. Widowed males also showed a higher rate for chronic conditions.

Our data are not adequate for further analysis; however, the general impression is that apparently no great difference exists between the illness patterns of the married and widowed.

III. Comparison of Married and Never Married Groups.—

Rates of the married group were age-adjusted to the never married population by the direct method using the never married population as the standard. Although the median ages of women in the two groups did not differ greatly (married women were about one year older than women who had never married), the men in the married group had a median age almost five years higher than that of the never married men.

Table VI shows that married females had higher morbidity rates than the never married by every measure except days of stay and average length of stay in hospital, although the differences were not great. The lower number of hospital days and average days per admission could be explained by the fact that the married women may be cared for at home by their spouses or children. Also, days and bed days of disability and incidence of illness include obstetric cases, although the number of such cases is small.

The comparison of rates of married and never married males did not show a consistent pattern.

MORTALITY AND MORBIDITY COMPARED

As mentioned earlier, certain basic differences between the morbidity and mortality data limit the comparability of the rates for the various marital status groups. These include such factors as the different sources of data used in computing

mortality rates, the exclusion of institutionalized persons from the California Health Survey population, the probability of underreporting in the Survey, and the nature of the morbidity rates as compared to mortality rates. Comparison of morbidity and mortality rates for the married, divorced, widowed, and never married groups should be done with full cognizance of these limitations.

The various morbidity rates of the divorced group are generally higher than those of the married group. This pattern is similar to the mortality pattern. In both mortality and morbidity data the differences between rates for the married and divorced groups are substantial.

Although higher mortality rates are reported among the widowed than among the married (for both sexes), morbidity rates for the widowed vary, and no clear pattern is evident.

SUMMARY AND CONCLUSIONS

Morbidity of married persons of each sex (age 25-74) was compared to morbidity of divorced, widowed, and never married persons. A consistent pattern of higher illness rates, both incidence and prevalence, was found among divorced persons than among married persons. The differences were of sizable magnitude and prevailed when the rates were adjusted for age, income level, and race. The cause of this pattern of higher rates was not investigated; we were only in a position to establish the fact that there was a difference.

Comparison of the married and widowed groups was difficult because of the older age of widowed persons; however, when adjustment was made for age no consistent pattern was evident, so that our data indicate no great difference between the health patterns of the two groups.

Similarly, comparison of the never married with the married showed little difference between the two groups.

It is a temptation to generalize from the findings of this study, but caution must prevail. Cause and effect statements cannot be made. For example, illness may cause a divorce or a divorce may cause illness; the recorded data cannot tell us.

These analyses were necessarily limited by the fact that the California Health Survey was not designed specifically for the purpose of comparing marital groups. However, within the limitations, we have compared morbidity data with mortality data for these groups. Perhaps our findings may be a stimulus to further studies designed to evaluate the differences—or lack of differences—between health patterns of marital status groups.

Mary Elizabeth Laughlin, Associate Public Health Analyst, and Marguerite Augustine, Health Education Consultant, California State Department of Public Health, gave helpful advice during the preparation of this paper.

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TABLE A. POPULATION DISTRIBUTION BY MARITAL STATUS, SEX, AND AGE (25-74) IN THE CALIFORNIA HEALTH SURVEY

MARITAL STATUS	AGE GROUP BY SEX															
	TOTAL		25–34		35–44		45–54		55–59		60–64		65–69		70–74	
	MALE	FEMALE	MALE	FEMALE	MALE	FEMALE	MALE	FEMALE	MALE	FEMALE	MALE	FEMALE	MALE	FEMALE	MALE	FEMALE
Total†	8,806	9,474	2,301	2,649	2,344	2,508	1,882	1,885	762	788	613	698	540	565	*364*	*381*
Married	7,370	7,138	1,869	2,217	2,090	2,099	1,617	1,447	634	537	511	432	406	272	*243*	*134*
Widowed	228	1,043	15	37	8	79	29	185	36	128	32	189	49	220	*59*	*205*
Divorced	461	751	102	183	103	222	120	171	43	86	31	37	38	29	*24*	*23*
Never married	741	535	314	211	143	107	116	82	49	36	37	39	45	41	*37*	*19*

*Figures are subject to sampling variation.
†Unknown included in total.
Note: For the interview period May 1954-April 1955.
Figures in italics represent 100-499 persons in sample.
Source: State of California, Department of Public Health, Bureau of Chronic Diseases, California Health Survey.

TABLE B. PER CENT OF POPULATION BY RACE, SEX, MARITAL STATUS, AND AGE (25-74) IN THE CALIFORNIA HEALTH SURVEY

MARITAL STATUS	RACE BY SEX									
	MALE					FEMALE				
	TOTAL	WHITE	SPANISH SURNAME	NEGRO	OTHER	TOTAL	WHITE	SPANISH SURNAME	NEGRO	OTHER
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
Married	83.7	84.6	79.7	77.1	71.3	75.3	76.3	72.3	61.7	78.0
Divorced	5.2	4.8	4.7	14.1	6.4	7.9	7.2	9.4	20.1	5.5
Widowed	2.6	2.5	3.2	4.0	3.2	11.0	10.8	10.2	14.8	11.0
Never married	8.4	8.1	12.4	4.8	19.1	5.7	5.6	8.1	3.4	5.5

Note: Percents are computed independently and may not add to totals.
Source: State of California, Department of Public Health, Bureau of Chronic Diseases, California Health Survey, 1954-1955.

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TABLE C. PER CENT OF POPULATION BY MARITAL STATUS, SEX, FAMILY INCOME, AND AGE (25-74) IN THE CALIFORNIA HEALTH SURVEY

MARITAL STATUS	FAMILY INCOME BY SEX									
	MALE					FEMALE				
	TOTAL	LESS THAN $2,000	$2,000-4,999	$5,000 AND OVER	UNKNOWN	TOTAL	LESS THAN $2,000	$2,000-4,999	$5,000 AND OVER	UNKNOWN
Total	100.0	10.3	39.6	45.7	4.3	100.0	14.1	39.3	42.0	4.6
Married	100.0	7.3	39.2	49.1	4.3	100.0	7.7	38.8	49.1	4.4
Divorced	100.0	23.6	43.4	29.3	3.7	100.0	33.4	46.6	16.9	3.1
Widowed	100.0	34.2	35.5	24.6	5.7	100.0	38.9	34.1	20.3	6.6
Never married	100.0	24.6	42.8	28.7	3.9	100.0	24.1	46.9	23.7	5.2

Note: Percents are computed independently and may not add to totals.
Source: State of California, Department of Public Health, Bureau of Chronic Diseases, California Health Survey, 1954-1955.

TABLE D. PER CENT OF POPULATION BY MARITAL STATUS, SEX, HEALTH INSURANCE COVERAGE, AND AGE (25-74) IN THE CALIFORNIA HEALTH SURVEY

MARITAL STATUS	HEALTH INSURANCE COVERAGE BY SEX					
	MALE			FEMALE		
	TOTAL	SOME	NONE	TOTAL	SOME	NONE
Total	100.0	57.5	42.5	100.0	53.9	46.1
Married	100.0	60.5	39.5	100.0	57.3	42.7
Divorced	100.0	44.5	55.5	100.0	45.0	55.0
Widowed	100.0	37.3	62.7	100.0	35.6	64.4
Never married	100.0	41.7	58.3	100.0	58.1	41.9

Note: Percents are computed independently and may not add to totals.

Source: State of California, Department of Public Health Bureau of Chronic Diseases, California Health Survey, 1954-1955.

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